ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM

X.

CLINICAL AND EXPERIMENTAL EXAMINATIONS IN PATIENTS SUFFERING FROM MB. MENIÈRI

INCLUDING A STUDY OF THE PROBLEM OF BONECONDUCTION

BY

DIDA DEDERDING

VOL. I

HELSINGFORS 1929
MERCATORS TRYCKERI AKTIEBOLAG

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Konsistorium, den 21. November 1927.

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Introduction.

Morbus Menièri has always been an enigmatical disease. When we consider, that I t a r d as early as 1821 gave us an excellent description of its appearance, that M e n i è r e in his treatise in 1861 states that the semicircular canals, if not the whole labyrinth, are the seat of the trouble, that F r a n k l-H o c h w a r t in 1894 was the first to publish an article giving a good survey of the subject and that several investigators have since dealt with it, it seems remarkable, that we know so little as to its etiology and pathogenesis. The principal importance of former otological investigations in this domain is that a series of labyrinthal affections with special etiology have been excluded: infectious labyrinthal affections, leucemia, hemorrhages in the labyrinth, labyrinthal affections resulting from traumata, brain tumours and metabolic diseases such as diabetes, anemia and nephritis. One of the causes of this imperfect knowledge of mb. Menièri is that it is not a fatal disease. No post mortem examination of a Menière-patient worth using exists. M e n i è r e's historical case of the girl, who became deaf a few hours after having riden on the top of a dilegence and soon after died, is both from a clinical and from a pathological-anatomical point of view only imperfectly described. Indeed there even exist two different descriptions from M e n i è r e's hand of the same case, one in a treatise from 1861 [1] and one in his translation of Kramer's otology from 1848.[2] The original description does not mention the existence of giddiness; this important symptom is first spoken of 13 years later. Various particulars, especially of pathological-anatomical nature, are also first given in the later description. It is no use now discussing what really was found in this post-mortem examination, but this incomplete description has

been the cause of our so long having held to the hypothesis that labyrinthal hemorrhage is a cause of mb. Menièri, even if several other theories have from time to time been broached, tending to show, amongst other things, that mb. Menièri may possibly be due to middle ear affections, oedema of the labyrinth, arterial hypertension, vasomotor disturbances or nuclear affections in medulla oblongata.

We will for the present use the term mb. Menièri as designating a disease, which, as far as the ear is concerned, shows itself by a varying function both of the acoustic and static part, and with regard to which it has been possible to exclude any specific etiology (such as syphilis, parotitis, encephalitis, tumors, diabetes, nephritis, professional disease of the internal ear or disseminated sclerosis).

The symptoms of this affection are, as far as the ear is concerned, partly subjective: giddiness, buzzings and deafness, and partly objective: acoustic and vestibular abnormalities found on detailed examinations of the function.

The problem of mb. Menièri has, for several years, occupied the attention of the Mun. Hosp.'s ear department. In 1924 S. H. Mygind[1] pointed out, that a series of cases with favourable prognosis exhibited such sudden and violent changes of hearing, that these changes could only be explained by the presence of corresponding changes in the endolabyrinthal pressure, for which reason he formulated the hypothesis of a »hypertensio auris internæ intermittens».

In pursuance of these ideas, the author has undertaken the task of investigating what conclusions a detailed examination of the acoustic function permit with regard to the causative changes in the ear in cases of this disease.

The author has endeavoured to attain her object partly by following up the spontaneous changes in these patients' hearing by means of numerous examinations, partly by determining the changes of hearing which can be experimentally produced.

On several points it has been necessary to examine the conditions in other ear affections and in normal individuals in order to come to an understanding of what is at the back of the conditions we encounter in the different acoustic tests.

The author has by these means come to results, which on certain points differ somewhat from prevailing theories, especially with regard to boneconduction.

Material and methods of investigation.

The present work rests upon examinations of 135 patients.

They have all been examined and treated in the ear department of the Municipal Hospital of Copenhagen, where 110 were in-patients and 25 out-patients.

23 of the in-patients had been transferred from the nerve department, where they had been admitted under a neurological diagnosis. 3 had been transferred from medical departments.

There are 70 males and 65 females from 14 to 74 years of age.

In the great majority of these patients the disease had shown itself several years before they were examined by us. In some of these cases it could be stated, that the disease had manifested itself about the patient's 13th or 14th year (Nos. 8, 18, 29, 117), in others that it already existed when the pt. was about 20 (Nos 4, 13, 76). In altogether 24 cases the disease is proved to have commenced before the pt.'s 25 th year. In many of the cases the observations extend over periods of 10 years.

The author has personally (since May 1926) been able to examine all the cases several times and the majority regularly over a period of from one year to eighteen months, some few even longer.

Many of the pts have been examined acoustically daily or several times a-day.

The pts have all been submitted to a general medical examination and in cases where a special neurological examination was required this has been performed by the neurological department. Wassermann reaction in the blood was performed in all cases, except No. 107. Hemoglobin and blood pressure were determined in all cases.

Lumbal puncture with ensuing cytological and chemical examination and Wassermann reaction was performed in 60 cases. In 42 pts ophthalmoscopy has been performed.

In 89 cases accounts have been kept as to the daily intake of fluids, diuresis and weight. Previous to a physio-theurapeutical

treatment all the in-patients were also examined at the physiotherapeutical clinic.

As far as it has been possible to do so, all measurements made with the tuning fork have been taken in one and the same room. The hearing of whisper has been taken in an underground passage 22 meters long.

Clinical examinations of the acoustic function in patients with Mb. Menièri.

General remarks. A reliable result of testing the acoustic function, as is well known, depends upon the observance of certain precautions.

Of these the most important for us are the following:

The same person must examine the patient at each examination.

The tuning fork must be struck and held in the same way at each examination.

At each determination of airconduction the tuning fork must be held close in front of the ear without touching it.

Each test must rest upon several determinations.

The examiner must ascertain that the pt. is sufficiently intelligent and musical to be able to perceive whether the fork has been struck or not, which is far from always being the case. The presumedly sound ear should — as several, especially E. Bloch[1] have pointed out — be examined at each examination, this being an excellent method of controlling the pt.'s statements as to the diseased side, especially if the other ear is in reality normal.

Only when these precautions have been observed can the figures be used for comparison with previous examinations of the pt., but they can only be used with reservation for comparison with those found in other pts., even if the same person has made all the examinations; this holds good especially in examinations as to boneconduction. Although these rules would seem to be matters of course, they are but too often neglected.

It must also be borne in mind when comparing boneconduction in different pts., that the pneumatisation of proc. mast., and consequently boneconduction, is different in different individuals.

as has been proved by Iwanoff, Okuneff, Mader, Frey.[1] Besides, the individual thickness of the skin and bones and the relation of dura to the skull may play some part (Wanner).

In examining boneconduction we must also remember, that what the pt. states he hears with the ear in question may in reality be due to transfer to the other ear. This question has been investigated by Frey[2] and by Blegvad[1], who in consideration of transfer to the other ear thinks that it is just as well to use the method, recommended by Schwabach[1], of placing the handle of the tuning fork on the vertex instead of on processus mastoideus.

The only way to make sure against transfer is to ask the pt. on which ear he hears the sound; he will then often of himself be aware that it is a case of transfer. When placing the tuning fork on processus mastoideus it is necessary, as mentioned above, to place it each time on the same spot; the importance of this has been proved by Bonnafont and V. Urbantschitsch[1] among others. The fork must not touch the cartilage.

We will now pass in review each of the usually employed acoustic tests with the results we have obtained from them.

Hearing of whisper and conversation. The importance of always having the same observer has to be especially considered in testing the hearing of whisper, as no two persons whisper at the same pitch and with the same power and distinctness. The number whispered must also be taken into consideration, as has been pointed out by Oscar Wolff[1] among others. All numbers that have a hissing sound (in English for instance 67, 56) have been in our material more easily caught than low figures (for inst. 22, 48). Numbers are sooner heard than sentences, as is also the case in other forms of deafness.

It is necessary to examine the hearing of whisper at a distance of at least 20 meters, as otherwise a slight diminuition of the hearing of whisper will escape notice.

No less than the half part of our pts namely, at periods hear whisper at a distance of 8—10—12 meters, although airconduction at the same time is shortened, boneconduction altered and the lower limit elevated. It is therefore evident that one cannot rest satisfied with an examination at a distance of about 7—10 meters, and, should the pt. hear at that distance, conclude that hearing of whisper is normal, as, for instance Wanner and Frey[3] have

done, what Herzog[1], therefore, has justly criticised. Indeed Frey[3] (l. c. page 894) goes still further, and says that with a hearing of whisper at a distance of 7—8 meters the possibility of more considerable pathological changes may be excluded.

One cannot deduce that hearing is normal even if whisper of numbers can be heard at a distance of 20 meters, before having substantiated that lower limit, upper limit, airconduction and boneconduction are normal, which is in no wise necessarily the case, as has been previously proved in pts with syphilis by Hasslauer, R. Lund[1] and O. Beck[1] amongst others. Siebenmann[4] states, that hearing of whisper may be 20 meters with abnormal boneconduction and lowered upper limit.

One cannot conclude, because pt. hears whisper at a distance of 20 meters, that his hearing of whisper is normal, as the normal, according to Morsak's[1] statements, may lie at about 89 meters.

In Menière-pts the hearing of whisper will often be found at 20 meters together with considerably shortened (for inst. Nos 1, 2, 37, 76, 78, 84, 95, 96, 106, 129), indeed even annihilated boneconduction (Nos 14, 95) or shortened airconduction and raised lower limit (Nos 20, 45, 61, 74, 76) or in a few cases lowered upper limit (No. 29: Ls. 9000, No. 49: Ls. 11000, No. 60: Ls. 12000, No. 112: Ls. 12000). A normal result of a single test does not therefore necessarily tell us anything. Suspicions should, however, always arise as to the possibility of pathological conditions, when a single test constantly gives abnormal values.

We do not therefore consider such a conclusion allowable, as that drawn, for inst. by Blumenthal in his investigations of boneconduction in pts. with shot wounds, when he, after having found boneconduction shortened in 84 % of the cases, whilst the result of the remainder of the acoustic examinations was normal, concludes that nothing was wrong with the ear and that the shortening of boneconduction was of »functional» nature. Hammerschlag also thinks, that a »disproportionally» shortened boneconduction with hearing of whisper at 5—6 meters must be considered as a symptom of hysteria.

When the pt. is unable to hear whisper, conversation must be employed and here the same consideration must be taken as to choice of numbers.

Pts suffering from mb. Menièri sometimes hear whisper as well or even better, than conversation, which is perhaps owing to the pitch of the speaking voice being too deep for them to hear.

Whisper and conversation, although they are imperfect methods of examination owing to the circumstance that there is no certainty of the same degree of strength and distinctness being used, possess an advantage over tuning fork examinations, as they are the sole acoustic tests in which it is possible to control that the pt. does not hear worse than he states.

The diminuition of hearing exhibited all stages, from the minimal degree with 20 meters whisper (Nos 16, 35, 93, 97, 102, 108, 123, 128, 132) in which it was only possible by repeated tuning fork examinations to prove a slight deviation from the normal, to complete, (Nos 8, 30, 79, 109) or practically complete, deafness (Nos 2, 39, 42, 72, 88).

The hearing of whisper was in half part of the cases very feeble: (ɔ: aa— 0,50) or extinguished.

In our pts the hearing of whisper and conversation was extremely fluctuating. The hearing of whisper could in the course of a few days increase to the double or more. The following are examples.

No. 2	19/6 28	v $\frac{0,05}{aa}$	27/6 28	v $\frac{20,00}{aa}$
No. 14	20/10 27	v $\frac{0,25}{0,75}$	20/10 27	v $\frac{8,00}{20,00}$
No. 20	4/3 27	v $\frac{5,00}{}$	13/3 27	v $\frac{20,00}{}$
No. 34	13/6 27	v $\frac{4,00}{2,00}$	16/6 27	v $\frac{20,00}{20,00}$
No. 37	15/10 26	v $\frac{5,00}{3,00}$	29/10 26	v $\frac{5,00}{20,00}$
No. 43	11/6 27	v $\frac{}{aa - 0,30}$	16/6 27	v $\frac{}{20,00}$
No. 45	19/7 26	v $\frac{4,00}{}$	23/8 26	v $\frac{20,00}{}$
No. 51	31/8 26	v $\frac{0,75}{0,05}$	14/10 26	v $\frac{3,00}{0,05}$
No. 56	9/5 27	v $\frac{}{0,25}$	14/6 27	v $\frac{}{12,00}$

No. 57	24/9	24	v $\frac{-}{aa}$	29/10	24	v $\frac{-}{20,00}$
No. 66	29/11	26	v $\frac{aa—1,00}{-}$	13/12	26	v $\frac{10,00}{-}$
No. 110	10/10	27	v $\frac{0,20}{0}$	29/10	27	v $\frac{0,40}{1,00}$
No. 113	31/1	28	v $\frac{2,00—3,00}{-}$	1/2	28	v $\frac{10,00}{-}$
No. 129	5/3	28	v $\frac{1,00}{3,00}$	10/3	28	v $\frac{20,00}{20,00}$
No. 131	6/6	28	v $\frac{3,00}{3,00}$	30/6	28	v $\frac{20,00}{6,00}$

Fluctuations may show themselves even in cases where the hearing of whisper lies at 20 meters, the pt. stating that he hears more or less dinstinctly alternately on the one or the other ear. These fluctuations would, in all probability, be still more distinct if one had a longer room at one's disposal. The following will show that fluctuations of hearing may be found in these cases by tuning fork examinations.

Hearing can, however, decrease as quickly as it increases as the following examples will show:

No. 2	9/6	28	v $\frac{12,00}{-}$	11/6	28	v $\frac{2,00}{-}$
No. 3	29/8	26	v $\frac{20,00}{20,00}$	1/9	26	v $\frac{1,00}{1,25}$
No. 8	2/12	26	v $\frac{3,00}{-}$	13/12	26	v $\frac{0,20}{-}$
No. 14	22/4	26	v $\frac{10,00}{-}$	24/4	26	v $\frac{0,50}{-}$
No. 20	6/5	27	v $\frac{20,00}{-}$	10/6	27	v $\frac{0,50— 2,00}{-}$
No. 36	3/6	27	v $\frac{0,75— 1,50}{-}$	13/6	27	v $\frac{0,10}{-}$
No. 37	30/5	27	v $\frac{20,00}{20,00}$	22/6	27	v $\frac{4,00}{4,00—20,00}$
No. 43	22/6	27	v $\frac{-}{12,00}$	4/8	27	v $\frac{-}{0,75}$

No. 45	23/8 26	$v \frac{20,00}{\quad}$	6/9 26	$v \frac{1,50}{\quad}$
No. 54	5/8 27	$v \frac{20,00}{\quad}$	6/8 27	$v \frac{2,00}{\quad}$
No. 62	31/1 27	$v \frac{4,00}{\quad}$	11/2 27	$v \frac{1,00}{\quad}$
No. 66	15/10 26	$v \frac{12,00}{\quad}$	15/11 26	$v \frac{1,00}{\quad}$
No. 75	17/1 27	$v \frac{20,00}{20,00}$	16/3 27	$v \frac{aa}{1,00}$
No. 99	8/6 28	$v \frac{\quad}{6,00}$	22/6 28	$v \frac{-}{aa}$
No. 126	23/6 28	$v \frac{\quad}{8,00—20,00}$	30/6 28	$v \frac{\quad}{0,50}$
No. 134	27/6 28	$v \frac{20,00}{0,20}$	18/7 28	$v \frac{1,00}{0,05}$

Hearing may at one time increase or decrease evenly for a longer period, at another change apparently capriciously in the course of some few days or hours.

These changes in hearing may appear not only in the commencement of the disease, but also after it has existed for a longer time, no. 29 for inst. had had his affection for 50 yrs, no. 2 for more than 30 yrs, no. 8 for 25 yrs, no. 36 for about 17 yrs.

The unilateral, or principally unilateral nature of the trouble may perhaps be the explanation why pt. in some cases does not himself observe the fluctuations. Even a considerable diminuition of hearing can in such cases entirely escape notice. In one case a pt., and an intelligent pt. (No. 15), only complained of giddiness and headache and declared that there was nothing wrong with his hearing although the acoustic examination showed:

$$v \frac{\quad}{0,35} a_1 M\ (15) \frac{-}{3} a_1 A\ (60) \frac{-}{5}$$

It may happen, when both ears are affected, that the pt. notices that he can only hear in the telephone with the right ear and a few days later only with the left, although he believes that he hears ordinary conversation equally well with both ears.

The variations in hearing will, as long as the disease is in its beginning and the hearing but slightly affected, escape both the

pt.'s and his doctor's notice and only be revealed after repeated acoustic examinations.

This changing of the acoustic function will, as long as one does not understand that it is typical, often confuse one, as the pt.'s statements are looked upon as incorrect, or the capricious changes of hearing, which the pt. speaks of are regarded as the expression of a functional state.

Airconduction. Where not otherwise stated, airconduction has been determined with an a_1 tuning fork, with a period of vibration of 60″.

In cases where airconduction was long, the tuning fork was allowed to vibrate for a time before being held in front of the ear, so as not to tire pt. and make him state a too short period of vibration.

Airconduction was abridged in all our cases. It also varied greatly from the one examination to the other. Airconduction and hearing of whisper correspond on the whole with one another, a diminished hearing for whisper accompanying a corresponding abridged airconduction which again increases at the same time as hearing of whisper.

It is often difficult for the pts to state when they cease to hear the fork in cases where their eventual buzzings are either identical with the note given by the fork, or so violent that they almost quite drown the note. Some pts (Nos. 33, 87, 89) state, that they only perceived the sound of the fork as an increased buzzing and not as a note.

A method, which enables one to control the correctness of a pt.'s dubious statement, is to stop the fork without the pt.'s knowledge and to see whether he discovers it.

As the pt. makes the same mistake each time, and as the figures are only compared with those obtained at former examinations of the same pt., these difficulties are of but little importance, as long as one is sure of the existence of buzzings. This should therefore be noted at each examination.

Upper limit. This is as a rule ascertained with S t r u y k e n's monochord; in some cases S c h u l t z e's monochord is used (recognisable in the case books by the limits being denoted by vibration ziphers corresponding to the musical intervals).

Upper limit is but slightly affected in fresh cases of mb. Menièri. The more the disease progresses the more liable is upper limit to be lowered.

In nearly all our pts it was changeable in all stages of the disease, even in the most advanced cases. It changed especially much in Nos. 2, 4, 7, 10, 16, 17, 19, 21, 25, 26, 29, 34, 37, 42, 47, 51, 56, 57, 59, 61, 62, 63, 71, 74, 75, 76, 77, 99, 103, 109, a few of them, for inst. No. 17 from 6000 to 12000, No. 29 from 9000 to 15000 (r.) 9000 to 12000 (l.) No. 42 from 5000 to 10000, No. 51 from 8000 to 14000 (r.) from 10000 to 14000 (l.), No. 63 from 12400 to 17530, No. 99 from 9000 to 14000, No. 103 from 9000 to 15000 (r.) from 7000 to 12000 (l.). The change could take place in the course of a few days.

Besides fluctuating, upper limit exhibits a peculiar condition; it *sometimes sinks, whilst hearing of whisper increases, boneconduction increases and lower limit sinks,* for inst.

No. 3 29/7 26 v $\frac{1{,}50}{0{,}75}$ Li $\frac{28}{28}$ Ls $\frac{16552}{16552}$ 6/8 26 v $\frac{2{,}00}{1{,}75}$ Li $\frac{24}{24}$ Ls $\frac{13920}{12400}$

No. 47 13/6 27 v $\frac{3{,}00—10{,}00}{}$ Li — Ls $\frac{17000}{}$ 15/7 v $\frac{20{,}00}{}$ Li — Ls $\frac{14000}{}$

No. 48 3/5 26 v $\frac{0{,}10}{17{,}00}$ Li $\frac{32}{24}$ Ls $\frac{15000}{15000}$ 13/7 26 v $\frac{2{,}00}{20{,}00}$ Li $\frac{26}{26}$ Ls $\frac{11000}{12000}$

No. 51 4/7 27 v $\frac{0{,}30}{}$ Li — Ls $\frac{11000}{}$ 4/8 27 v $\frac{0{,}75}{}$ Li — Ls $\frac{9000}{}$

No. 53 4/5 27 v $\frac{1{,}00}{}$ Li — Ls $\frac{16000}{}$ 16/5 27 v $\frac{1{,}75}{}$ Li — Ls $\frac{14000}{}$

No. 53 24/4 27 v $\frac{0{,}10}{}$ Li — Ls $\frac{16000}{}$ 18/2 27 v $\frac{6{,}00}{}$ Li — Ls $\frac{15000}{}$

No. 56 9/5 27 v $\frac{}{0{,}25}$ Li $\frac{}{45}$ Ls $\frac{}{15000}$ 14/6 27 v $\frac{}{12{,}00}$ Li $\frac{}{24}$ Ls $\frac{}{14000}$

No. 76 22/3 27 v $\frac{}{10{,}00—20{,}00}$ Li $\frac{}{36}$ Ls $\frac{}{19500}$ 27/7 27 v $\frac{}{20{,}00}$ Li $\frac{}{26}$ Ls $\frac{}{16000}$

Corresponding to this there were cases where *upper limit rose when hearing of whisper decreased and lower limit ascended,* for inst.

No. 51 16/6 27 v $\frac{0{,}75}{}$ Li $\frac{24}{}$ Ls $\frac{8500}{}$ 28/6 27 v $\frac{0{,}40}{}$ Li $\frac{60}{}$ Ls $\frac{11000}{}$

No. 53 2/5 27 v $\frac{1{,}00}{}$ Li $\frac{22}{}$ Ls $\frac{14500}{}$ 4/5 27 v $\frac{1{,}00}{}$ Li $\frac{36}{}$ Ls $\frac{16000}{}$

This corresponds to the circumstance that in some of our pt.'s *we found upper limit best on the ear, which heard worst,* for inst.

No. 29 4/10 $v \frac{20,00}{0,15} a_1 M \frac{8}{3} a_1 A \frac{56}{19} Li \frac{17}{55} Ls \frac{9000}{12000}$

No. 29 11/10 $v \frac{20,00}{0,40} a_1 M \frac{16}{9} a_1 A \frac{63}{31} Li \frac{}{38} Ls \frac{10000}{12000}$

No. 34 9/8 $v \frac{20,00}{20,00} a_1 M \frac{9}{12} a_1 A \frac{35}{45} Li \frac{15}{15} Ls \frac{9000}{7000}$

No. 51 21/12 26 $v \frac{2,00}{0,05} a_1 M \frac{12}{9} a_1 A \frac{40}{4} Li \frac{21}{>150} Ls \frac{9000}{11000}$

No. 51 31/1 27 $v \frac{1,00}{0,05} a_1 M \frac{11}{5} a_1 A \frac{36}{0} Li \frac{21}{>150} Ls \frac{11000}{13000}$

The rise of upper limit together with sinking hearing may be met with in other ear troubles, for inst. syphilis (Rob. Lund[2]: No. 24 p. 210, No. 52, p. 213, No. 65, p. 217, No. 73, p. 220); Grahe[1] observes that in pts with nephritis upper limit sinks when hearing increases. None of the above mentioned authors, however, seem to be aware that this is a characteristic condition, although Zwaardemaker has already in 1895 drawn attention to this condition being characteristic for the initial stages of »sclerosis».

It is then not necessarily correct to conclude, as formerly, from a relatively high upper limit together with a relatively bad hearing, that upper limit is not influenced by the disease.

Lower limit. This was determined with Bezold's continuous scale. The determination of lower limit is, perhaps, the most difficult of all acoustic tests. It is often difficult to get a pt. to distinguish between hearing and the feeling of vibration of air. He feels something, and, in his eagerness, declares he hears a note. Even when following the method recommended by Saxtorph Stein, which is to let pt. close his eyes and state when he notices the tuning fork approaching, mistakes may happen. This may also be the case, when one further makes use of Blegvad's[2] method of striking the fork and then stopping it before it is held to the ear. None of these methods insure one against feeling contra hearing; for although the pt. states that he does not hear an unsounded fork, as he cannot feel it, one does not therefore know whether a sounded

fork is heard or felt. For instance 11 of our patients (Nos. 8, 10, 32, 41, 70, 82, 88, 92, 116, 117, 120) declare that they hear the fork if it is only quite gently waved in front of their ears without being sounded. In such cases the values given cannot of course be made use of. Politzer[1] also distinctly points out that, »Flattern vor dem Ohre als Tastempfindung leicht zu Täuschung Anlass giebt», when a fork under C_1 is used.

This is presumably what often has taken place when we in literature find the lower limit given as normal, in spite of the hearing of whisper being almost nil; at all events this was proved to be the explanation in corresponding cases of our material (Nos. 4, 8, 41, 70, 82, 88, 117).

The buzzings are often so violent that it is impossible to be clear as to pt.'s lower limit, because the low note of the fork coincides with the buzzings. The patients express this by saying: »The buzzings get louder», when the sounded fork is held up to the ear. This is again a reason for always inquiring at the same time as to buzzings and noting the reply.

In our pts lower limit was nearly always raised. It was also always changing, and often changed greatly.

(No. 14: 24—>150, No. 29: 16—150, No. 36: 21—>150 No. 42: 90—>150, No. 43: 24—90, No. 51: 15—65, No. 56: 15—60, No. 57: 40—90, No. 63: 19—55, No. 66: 26—90, No. 75: 28—90, No. 99: 24—>150, No. 121: 15—90, No. 127: 15—90).

These great fluctuations may take place in the course of a few days. The above figures show at the same time, that the reduction of lower limit may attain values otherwise met with in otosclerosis.

Lower limit keeps pace pretty well with hearing of whisper and airconduction, so that bad hearing of whisper is accompanied by raised lower limit and abridged airconduction; when hearing of whisper and airconduction improves, lower limit again rises.

Boneconduction. When not otherwise stated boneconduction, is tested with an a_1 fork with vibration period of 15″ for normal hearing.

With this test it is necessary to be sure that the pt. hears a true note, and does not only feel a whirring sensation. When boneconduction has almost disappeared, a dull humming is sometimes per-

ceived, as has been the case with some of our pts (Nos. 7, 14, 49, 51, 72). To insure against an eventual case of transfer, enquiry should be made as to in which ear pt. hears the tone. Nos. 5, 14, 28, 43, 74 and 119 often hears the note on the opposite side to that on which the fork was placed. It happened that the same patient could one day hear boneconduction »normally», and another day by transfer to the other ear.

It is hardly correct, in cases where such a transfer to the opposite side takes place, to quote boneconduction as nil, as the possibility of boneconduction on this side cannot be excluded. In these cases the duration of boneconduction was made a note of, also on which ear it was heard (for inst. $\frac{12}{8}$ ℌ : heard on right).

There is a great diversity in the timbre, even when pt. hears the note, the note the one day being clear and shrill, and on another dull and slightly humming, again the next time, perhaps, perceived only as humming, or indeed not perceived at all.

All variations of boneconduction values appeared in our material: normal, abridged and lengthened.

Many of the patients exhibited abridged boneconduction at the one examination and normal, or almost normal, at another.

No. 7: 9—16, No. 12: 6—16, No. 15: 4—14, No. 21: 8—16, No. 28: 5—16, No. 33: 0—15, No. 45: 7—15, No. 62: 7—15, No. 63: 6—15, No. 69: 6—14, No. 74: 6—14, No. 116: 4—15, No. 122: 8—16. In others the change was from abridged bone conduction to lengthened (No. 3: 5—19, No. 29: 3—22, No. 68: 8—17, No. 126: 9—21) or from normal to lengthened (No. 10: 15—22, No. 20: 15—25, No. 58: 15—23, No. 66: 14—20). The majority fluctuated from more to less abridged boneconduction (for inst. No. 2: 0—10, No. 14: 0—12, No. 15: 3—12, No. 34: 6—11, No. 36: 3—9, No. 43: 0—11, No. 51, 0—9, No. 53: 8—11, No. 87: 0—8, No. 95: 4—11). Thus boneconduction may change just as much as hearing of whisper and airconduction, and a single or only a few examinations can, therefore, never give a correct result.

In many patients a remarkable circumstance with respect to boneconduction was observed, namely that is was abridged, indeed had quite disappeared, together with a fully preserved hearing of whisper, and comparatively good airconduction

and lower limit, in some cases (Nos. 15, 35, 56, 108, 123) even with normal lower limit and airconduction only shortened by a few seconds.

As examples we give:

No. 1 14/3 28 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{5}{6}$ a_1A (40) $\frac{26}{19}$ Li $\frac{21}{24}$ Ls $\frac{16000}{17000}$

No. 2 27/6 28 v $\frac{20,00}{aa}$ a_1M (15) $\frac{7}{5}$ a_1A (60) $\frac{22}{15}$ Li $\frac{24}{42}$ Ls $\frac{17000}{9000}$

No. 14 29/8 26 v $\frac{14,00—20,00}{20,00}$ a_1M (15) $\frac{0}{0}$ a_1A (40) $\frac{12}{30}$ Li $\frac{40}{36}$

No. 15 15/12 26 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{11}{11}$ a_1A (60) $\frac{55}{55}$ Li $\frac{15}{15}$ Ls $\frac{18548}{18548}$

No. 35 11/5 27 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{9}{8}$ a_1A (60) $\frac{61}{55}$ Li $\frac{15}{15}$ Ls $\frac{18000}{18000}$

No. 36 30/7 25 v $\frac{0,35}{20,00}$ a_1M (10) $\frac{3}{5}$ a_1A (25) $\frac{0}{14}$ Li $\frac{45}{15}$ Ls $\frac{13140}{13140}$

No. 37 19/11 26 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{6}{6}$ a_1A (60) $\frac{30}{44}$ Li $\frac{17}{15}$ Ls $\frac{14000}{15000}$

No. 56 4/8" 27 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{15}{9}$ a_1A (60) $\frac{50}{50}$ Li $\frac{15}{15}$ Ls $\frac{16000}{15000}$

No. 78 29/6 27 v $\frac{< 20,00}{20,00}$ a_1M (15) $\frac{3\frac{1}{2}}{6}$ a_1A (60) $\frac{22}{31}$ Li $\frac{24}{20}$ Ls $\frac{17000}{17000}$

No. 84 20/8 27 v $\frac{20,00}{0,50}$ a_1M (15) $\frac{4}{1—2}$ a_1A (60) $\frac{25}{16}$ Li $\frac{19}{36}$ Ls $\frac{18000}{17000}$

No. 87 14/11 27 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{6}{7}$ a_1A (60) $\frac{22}{34}$ Li $\frac{24}{20}$ Ls $\frac{14000}{13000}$

No. 93 21/3 28 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{9}{9}$ a_1A (40) $\frac{32}{26}$ Li $\frac{15}{15}$ Ls $\frac{15000}{19000}$

No. 95 13/12 26 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{3}{0}$ a_1A (60) $\frac{29}{33}$ Li $\frac{24}{24}$ Ls $\frac{15624}{15624}$

No. 95 4/4 28 v $\frac{20,00}{20,00}$ a_1M (15) $\frac{0}{0}$ a_1A (40) $\frac{15}{15}$ Li $\frac{34}{34}$ Ls $\frac{16000}{16000}$

No. 96 13/1 28 v $\frac{20{,}00}{20{,}00}$ a_1M (15) $\frac{4}{4}$ a_1A (40) $\frac{18}{21}$ Li $\frac{24}{21}$ Ls $\frac{17000}{18000}$

No. 106 20/3 27 v $\frac{20{,}00}{0{,}10—2{,}50}$ a_1M (15) $\frac{6\frac{1}{2}}{5}$ a_1A (40) $\frac{23}{11}$ Li $\frac{15}{24}$ Ls $\frac{15500}{13000}$

No. 108 1/6 28 v $\frac{20{,}00}{20{,}00}$ a_1M (15) $\frac{8}{7}$ a_1A (40) $\frac{27}{35}$ Li $\frac{15}{15}$ Ls $\frac{18000}{17000}$

No. 122 19/3 28 v $\frac{20{,}00}{—}$ a_1M (15) $\frac{7\frac{1}{2}}{—}$ a_1A (40) $\frac{30}{—}$ Li $\frac{15}{—}$ Ls $\frac{16000}{—}$

No. 123 25/5 28 v $\frac{20{,}00}{20{,}00}$ a_1M (15) $\frac{10}{10}$ a_1A (40) $\frac{40}{30}$ Li $\frac{15}{15}$

No. 128 19/3 28 v $\frac{20{,}00}{20{,}00}$ a_1M (15) $\frac{10}{6}$ a_1A (40) $\frac{37}{26}$ Li $\frac{18}{20}$ Ls $\frac{16000}{15000}$

No. 129 8/3 28 v $\frac{10{,}00}{20{,}00}$ a_1M (15) $\frac{5}{2}$ a_1A (40) $\frac{18}{29}$ Li $\frac{15}{24}$ Ls $\frac{13000}{13000}$

There are besides numerous cases, where boneconduction must be considered as disproportionately abridged in relation to air-conduction, lower limit and hearing of whisper, for inst.:

No. 2 11/6 28 v $\frac{2{,}00}{aa}$ a_1M (15) $\frac{0}{0}$ a_1A (40) $\frac{13}{13}$ Li $\frac{36}{130}$ Ls $\frac{15000}{7500}$

No. 3 6/8 28 v $\frac{—}{0{,}30}$ a_1M (15) $\frac{—}{0}$ a_1A (25) $\frac{—}{15}$ Li $\frac{—}{22}$ Ls $\frac{—}{13920}$

No. 8 13/11 26 v $\frac{1{,}25}{—}$ a_1M (15) $\frac{3}{—}$ a_1A (60) $\frac{24}{—}$ Li $\frac{24}{—}$ Ls $\frac{14000}{—}$

No. 12 16/8 27 v $\frac{20{,}00}{0{,}2—6{,}00}$ a_1M (15) $\frac{14}{0}$ a_1A (60) $\frac{45}{21}$ Li $\frac{16}{45}$ Ls $\frac{18000}{16000}$

No. 30 10/3 27 v $\frac{0{,}50}{0}$ a_1M (15) $\frac{0}{0}$ a_1A (60) $\frac{18}{0}$ Li $\frac{24}{—}$ Ls $\frac{13000}{—}$

No. 46 18/10 26 v $\frac{4{,}00}{4{,}00}$ a_1M (15) $\frac{5\frac{1}{2}}{4}$ a_1A (60) $\frac{47}{45}$ Li $\frac{20}{21}$ Ls $\frac{13000}{13000}$

No. 56 9/7 26 v $\frac{20{,}00}{6{,}00—12{,}00}$ a_1M (15) $\frac{9}{5}$ a_1A (60) $\frac{45}{40}$ Li $\frac{15}{22}$ Ls $\frac{16000}{14000}$

No. 72 4/6 27 v $\frac{0}{0{,}20—0{,}40}$ a_1M (15) $\frac{0}{0}$ a_1A (60) $\frac{8}{24}$ Li $\frac{>150}{30}$ Ls $\frac{6000}{14000}$

No. 83 28/10 27 v $\frac{20{,}00}{6{,}00}$ a_1M (15) $\frac{8}{3}$ a_1A (60) $\frac{42}{30}$ Li $\frac{15}{21}$ Ls $\frac{16000}{16000}$

No. 86 24/10 27 v $\frac{0{,}30}{0{,}20}$ a_1M (15) $\frac{2}{0}$ a_1A (60) $\frac{17}{16}$ Li $\frac{24}{30}$ Ls $\frac{10000}{11000}$

No. 87 18/11 27 v $\frac{3{,}00}{3{,}00}$ a_1M (15) $\frac{0}{0}$ a_1A (60) $\frac{22}{29}$ Li $\frac{32}{26}$ Ls $\frac{16000}{11000}$

Many Menière-pts often hear badly in the telephone although their hearing of whisper is fairly good. This is probably owing to their boneconduction being often bad. In pts with otosclerosis namely, we on the opposite often find, that they hear well in the telephone as compared to their hearing of ordinary conversation, this being caused by their good boneconduction.

Rinnes' test was most often shortened positive, in some it was however negative, for inst. Nos. 8, 26, 38, 41, 50, 51, 64, 66, 67, 70, 82, 110, 111; it could also vary in one and the same pt. from + to ÷, for inst. Nos. 3, 14, 18, 36, 45, 51, 55, 84.

The acoustic function as a whole. Like former authors, we have found that hearing of whisper, airconduction, boneconduction, lower and upper limit are affected in pts with mb. Menièri.

We have also shown that in the majority of cases the hearing fluctuates greatly, irrespective of the duration of the disease.

The very changing picture presented in some measure follows fixed rules: bad hearing of whisper corresponding to short airconduction and elevated lower limit, boneconduction, as a rule at the same time becoming shorter. Good hearing of whisper will, as a rule, be accompanied by long airconduction, low lower limit and, most frequently, longer boneconduction. The factors are not always perfectly paralel.

It has therefore been necessary, in order to explain this changing picture, to undertake numerous acoustic examinations. With some few pts (Nos. 3 and 14) we have made from 50—100 series of acoustic examinations besides separate tests, for inst. for whisper alone. No. 14 was examined daily for a period extending over 18 months, often many times a-day; the case book gives examinations for a period of 28 months. Out of consideration to their length, none of the case books include all the acoustic examinations.

Buzzings and other subjective acoustic phenomena. Buzzings were observed in 115 pts, in 64 of these on both ears and in 51 only

on the one. In 20 (Nos. 22, 37, 40, 58, 66, 71, 80, 93, 97, 98, 103, 105, 108, 110, 111, 123, 125, 128, 133, 135) no buzzings have ever been present; these cases, however, differed in no other wise from the other Menière pts.

In the 51 cases of unilateral buzzings, the acoustic affection was also unilateral in 12 only (Nos. 13, 29, 33, 45, 54, 56, 58, 73, 76, 85, 89, 91). In 3 cases (Nos. 32, 117, 120) the statements of the patients at the acoustic examination were too inexact to be relied upon. In the remaining 36 cases the acoustic affection was bilateral. In 9 of these patients (Nos. 10, 18, 47, 48, 49, 90, 94, 106, 112) the reduction of hearing was considerable on both sides, while on the better side in 10 cases (Nos. 21, 43, 46, 50, 57, 99, 100, 107, 121, 134) it was pronounced, and in the remainder 17 cases (Nos. 12, 15, 19, 20, 27, 36, 52, 53, 55, 61, 65, 69, 70, 74, 113, 115, 122) but slight.

Buzzings may then be totally absent in mb. Menièri, and also unilateral in spite of otherwise distinct bilateral acoustic affection.

The intensity of the buzzings varied greatly; they even disappeared entirely for hours and even days.

In some cases they were so slight, that the pt. only noticed them when everything was perfectly quiet, and especially at night. In others the buzzings are extremely troublesome, forming, indeed, the pt.'s chief complaint. In a number they were worst in the morning, when the patient woke, decreasing in the course of the day and disappearing quite in the evening.

But a very few pts were able to explain what it was that made the buzzings alter. Nos. 1, 2, 3, 7, 11, 18, 28, 32, 36, 41, 42, 43, 53, 54, 55, 65, 83, 85 had the worst buzzings during their attacks of giddiness. In No. 61 the buzzings ceased at the beginning of the attack, while No. 10 first felt buzzings at the end of the attack. In (Nos. 21, 24, 28, 35, 38, 39, 52, 56, 66, 70, 76) buzzings appeared during some attacks but not during others.

From this it appears, that buzzings are a frequent, but not an absolutely characteristic symptom of mb. Menièri.

Some pts had *autophonia*, for inst. Nos. 9, 14, 58, 78 and some *diplacusis*, for inst. Nos. 7, 14, 17, 107. This phenomena is doubtless a more frequent one than appears from the case books, as the pts were not systematically questioned on this point.

Relation between hearing and giddiness. The vestibular phenomena lie outside the scope of the present work.*

We will here only discuss the relation between giddiness and hearing.

It appears that giddiness and a feeling of insecurity often accompany the greater fluctuations in hearing. 17 pts (Nos. 1, 4, 8, 13, 14, 18, 29, 43, 50, 53, 54, 69, 72, 90, 99, 104, 134) declared that their hearing always grew better in sympathy with the attack, i. e. even during the same. 9 pts (Nos. 20, 32, 41, 45, 65, 76, 82, 90, 91) stated, on the contrary, that their hearing got worse during the attacks. A few (Nos. 36, 37, 89) stated that their hearing remained unaltered. The remainder were unable to give any information as to the relation of the fluctuation of hearing to attacks, which

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* These will be treated of by S. H. Mygind in a special work from which we quote the following:

Nystagmus abnormalities were found in all the pts. observed. 39 pts. were observed during severe attacks of giddiness, many during less pronounced attacks.

In a small group of 9 cases the postrotatory nystagmus exhibited so considerable a reduction, that it might be looked upon as an expression of a reduced function. The caloric nystagmus was absent in only one of these cases, in the remainder it was, as a rule, feeble with, eventually, a defective reversal on forward bending. 5 of these pts. exhibited spontaneous nystagmus to the relatively sound side, often accompanied by a component directed vertically downwards.

The nystagmus phenomena in all the remaining 126 cases were of much slighter character. The postrotatory nystagmus was either without pathological difference or lengthened to the one side, corresponding frequently to a spontaneous or latent spontaneous nystagmus. The caloric nystagmus in this group exhibited no abnormality beyond an abnormal reversal in a few cases.

All the pts. in this group with the exception of 12, exhibited spontaneous nystagmus. This was, as a rule, horizontally-rotatory. A vertical component, almost always pointing downwards, was also often observed. This vertical nystagmus was at times predominant or the only one present, at other times the nystagmus was more or less purely rotatory or purely horizontal. In only four cases (17, 18, 129, 132) was there a spontaneous nystagmus beating at the same time horizontally to the one, and rotatorially to the other side. The spontaneous nystagmus beat twice as often to the acoustically most affected ear as to the opposite one, without it being possible to refer the direction of the nystagmus to a relatively good acoustic function in the one, or a relatively bad in the other case. The nystagmus, in several pts. was changeable, that is sometimes to the right and sometimes to the left. More or less typical otolith phenomena were frequent. Pathological differences by counterrolling were often observed. Nos. 2, 6, 14, 53, 58, 85, 88 exhibited distinct pseudofisthel symptoms. Inhalation of amylnitrite (tried on 70 pts.) gave a distinct reaction in about ¾ of the cases. In some of these the nystagmus had one direction during the hyperemic phase and an opposite during the following anemic phase.

is partially due to their defective observation of their disease, as they are so confused during the attacks that they are unable to heed anything, and afterwards so tired that they cannot observe themselves properly. It may also be, that hearing on the one ear is so good, that the imperfect function of the other escapes observation. We have, however, by examining such pts immediately before and after an attack, been able sometimes to substantiate that, unknown to themselves, fluctuations in hearing do take place in connection with the attacks. In No. 29 (4/10, 27) a rise of hearing was found. In No. 43 and 66 a rise of hearing was once observed, at anothertime a fall. There is, then, no rule as to how the hearing is affected by an attack; one can only say that it most often undergoes a change. In bad periods, by which we understand such as exhibit frequent attacks of giddiness or uncertainty and considerable buzzings, the hearing is particularly fluctuating and may change in a single hour from v $\frac{}{0{,}30}$ to $\frac{}{20{,}00}$, as we have observed in connection with an apoplectiform attack (No. 14).

From our material it appears, that there is generally but little giddiness during periods of constantly reduced hearing. The same holds good in periods where hearing is good. Giddiness, thus shows itself especially when hearing is fluctuating: the more the hearing changes, the worse the patients feels, which is not only evinced by attacks of giddiness and unsteadiness, but also by buzzings and pressure in the ears.

Unsteadiness and smaller attacks are more frequent during periods of bad hearing than during periods of good hearing. Several smaller attacks often compensate for a greater attack. A great attack is frequently succeded by a lengthy good period.

Brief attacks of giddiness exactly ressemble those caused by catheterization, as we have also seen in several of our patients. (Nos., 6, 12, 13, 14, 19, 27, 38, 41, 55, 57, 62, 66, 70, 82, 88).

In some pts (Nos. 2, 6, 14, 53, 58, 85, 88) distinct nystagmus could be produced by compression and aspiration in the external meatus. No. 100 and 132 felt giddy in bad periods, when the ear was only touched. No. 7 had so violent an attack of giddiness, when he sneezed that he fell off his bicycle and came to himself in the gutter. Nos. 2 and 27 had attacks of giddiness

when they blew their noses. Some of our pts (Nos. 14, 62, 74) felt »sea sick» during change of pressure, such as produced in the so-called bell experiments to be later mentioned, and that although there is here only question of a comparatively slight and evenly increasing and decreasing change of pressure.

In a single pt. we observed about 40 attacks, during the great majority of which hearing improved in direct correspondance with the attack.

The general idea, that hearing always grows worse after an attack is then, incorrect. The one time after the other pts, for inst. (Nos. 8, 54, 72), said, that if only an attack would come on, the pressure on the ears would cease and their hearing improve.

An increasing hearing in connection with an attack has been observed by Morsak[2] and Lermoyez (»vertige qui fait entendre») and Lannois and Chavanne. G. Heermann[1] also relates several cases of rise of hearing, among others one where v was 0,10 before the attack, and 6,00 immediately after it.

The otoscopical picture and the condition of tuba. In 98 of our pts bilateral changes of the tympanic membrane were met with, unilateral in 14. Of these 6 (Nos. 44, 52, 80, 89, 112, 129) exhibited abnormal left membrane, 8 (Nos. 19, 29, 56, 101, 108, 115, 120, 126) abnormal right membrane. Only 23 had normal tympanic membranes on both sides. (Nos. 1, 2, 18, 19, 22, 23, 45, 48, 73, 76, 85, 90, 91, 92, 97, 102, 104, 109, 120, 123, 130, 131, 132).

In a good half part of the cases the change consisted in retractions, varying from quite minimal to maximal. In no less than 47 (Nos. 3, 4, 5, 9, 12, 15, 18, 25, 27, 28, 31, 32, 34, 38, 39, 42, 43, 44, 46, 47, 49, 50, 51, 53, 55, 58, 60, 61, 62, 64, 65, 66, 67, 71, 74, 78, 79, 86, 87, 94, 99, 100, 103, 105, 110, 117, 119) these retractions were remarkably pronounced.

Besides this, defective light reflex was often observed or the membrane was thickened, fibrously changed, now and then exhibiting chalky deposits. In other cases again the membrane was atrophied or abnormally translucent, with remarkably distinct articulatio incudo-stapedia. In many the manubrium mallei projected reliefwise. Dry perforation was only seen in 3 cases (Nos. 10, 24, 127). On account of their frequency these abnormalities

of the tympanic membrane in our pts cannot be passed over as being accidental, all the more so as the most pronounced changes were especially observed in such pts as exhibited the most defective acoustic function, while on the contrary a normal otoscopy on both sides was especially observed together with slight acoustic changes.

It must also be pointed out that of 23 such cases with bilateral normal otoscopy the 11 represented quite fresh ear phenomena (Nos. 1, 19, 73, 76, 85, 90, 91, 97, 102, 104, 123). Indeed No. 97 and 123 had not even any buzzings.

Of the 14 pts with unilateral abnormalities of the tympanic membrane the 4 had a corresponding unilateral acoustic trouble (Nos. 29, 56, 89, 126). The remaining 10 had, indeed, a bilateral disease, but of such nature that the acoustic changes on the otoscopically normal side were quite slight. Now, allthough every change in the tympanic membrane is synonymous with a more or less pathologically altered middle ear, a normal otoscopy on the other hand, does not necessarily involve a normal state of the middle ear.

The cause of these abnormalities of the tympanic membrane are possibly many. Resorption of air in a closed middle ear is generally looked upon as the principal cause of retraction. If this is correct, the same process as has acted on the tympanic membrane must also have been acting on the fenestrae, possibly with a further action upon the contents of the labyrinth.

It was, therefore, in this connection of importance to investigate in how far tubal stenoses were to be found, in this material, as a cause of the supposed closure of the middle ear.

Only 79 pts were examined for tubal stenosis, catheterization purposely not being performed on the remainder in order not to influence the results of other treatments. In no less than 43 bilateral tubal stenosis was found, unilateral in 13. In 23 no stenosis was proved, which however in no wise means that stenosis may not have been present at certain periods, as the stenosis in all our patients was of a distinctly intermittent nature. In some of the pts (Nos. 3, 4, 14, 16, 17, 24, 55, 70) the stenosis was, at times, so obstinate that bougies had to be used.

In this connection we may mention, that in the 50 cases of mb. Menièri published by G. Heermann[1, 2], retraction was

observed in some, and normal tympanic membrane in others. Rohrer[1] has published 11 cases of »Torpor nervi acustici», of which 5 had attacks of giddiness, all exhibiting deafness and abolition of boneconduction; in all of them the tympanic membrane was »swollen» and there was stenosis of the tuba. Stetter has 3 cases of mb. Menièri, in all of them the tympanic membrane was thickened, Gerber a case with retracted tympanic membrane, and Stephen Mackenzie a case with retracted tympanic membrane and tubal stenosis on left side. Gradenigo[1,2], Gellé[3], Löwenberg, Politzer[2] and Burnett think that otitis interna generally, must have a relation to the middle ear, and Gradenigo[2] also often finds that the so called »sound side» exhibits changes in the middle ear. Wittmaack[2] considers that the middle ear is the primary seat of trouble in mb. Menièri.

Thornval[1,2] on the contrary states that the tympanic membranes are normal in mb. Menièri and has never at any period in the disease seen signs of middle ear trouble. There are however in the 8 case books, published in his work[1] 5 pts with changes of the tympanic membrane.

Sensation of pressure. 53 pts suffered, particularly in bad periods, from a pressure in their ears, varying from a sensation of a flap over the ear to a real pain, often localized to the jaw joint (f. inst. Nos. 14, 51, 69).*

Relation between uni- and bilateral affection. Bilateral acoustic abnormalities were found, in the great majority of our pts namely in 107 of 135. In 17 cases only a unilateral acoustic disease could be proved, of these 8 were on the right (Nos. 13, 29, 33, 56, 76, 85, 90, 126) and 9 on the left side (Nos. 36, 45, 54, 58, 66. 73, 89, 91, 92), whilst in Nos. 32, 120 hearing could not be determined with tuning forks, as pts stated that they also heard forks when not sounded, both by airconduction and by boneconduction, also by testing of lower limit, whilst hearing of whisper was 20 m. on either side.

The affection, from an acoustic point of view, was, in about the half of the cases, almost the same on both sides, that is as far

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* A tubal stenosis may often be present, but does not seem to be an indispensable condition to this phenomenon.

as it is possible to draw comparisons here, where hearing often varies in different degrees on the two ears.

In 9 cases hearing was »practically» speaking normal on both sides (Nos. 16, 35, 93, 97, 102, 108, 123, 128, 132), of these 4 had bilateral buzzings (Nos. 16, 35, 102, 132), whilst the remaining 5 had no buzzings.

The acoustic function of the remaining pts generally exhibited a considerable difference between the two sides. In several cases the apparently »sound ear» was first proved to be affected after frequent and accurate examinations, because, as previously pointed out, hearing of whisper at 20 meters is not sufficient for the decision of normal hearing.

In cases where only a single test gave a slight abnormality on the »sound» side, we have not accounted the affection as bilateral, because allowance must always be made for errors on the part of both investigator and pt. In cases, however, where the abnormality has been proved several times, even only by some of the tests, for inst. boneconduction, airconduction and upper limit, we have deemed ourselves justified in reckoning the affection as of bilateral nature.

In some cases, single tests gave such slight abnormalities, that we did not feel ourselves justified in including them among bilateral affections. It does not therefore follow as a matter of course that these cases may not later on reveal themselves as bilateral. Several of them (Nos. 54, 73, 76, 85, 90, 91, 92, 126) are so fresh, that the acoustic change is still quite slight, even on the diseased side*. An investigation of the history of bilateral cases shows, that the deafness has at first, even for several years, been apparently unilateral, (Nos. 2, 40). It is first later on that the deafness attains such a degree that the pt. notices it (for inst. Nos. 2, 4, 8, 18, 38, 72, 79, 82, 110).

On comparing the acoustic results with the otoscopy, which latter, as previously stated, shows the majority of pts as having bilateral abnormalities, we find, that of the 17 cases with unilateral acoustic affections 12 exhibited normal and 5 (Nos. 13, 33, 36, 54, 58) only minimal changes. There were changes of the tympanic

* During the printing of the book No 73 has turned out as a pronounced bilateral case $\left(v\frac{0}{4{,}00}\right)$.

membrane on the acoustically affected side in 10 cases and no changes in 7. Thus the otoscopical and the acoustical phenomena seem to be in satisfactory accordance; both of them showing a pronounced tendency to be bilateral.

The fact, that — as formerly mentioned — more than half the pts declared that the buzzings were bilateral, indicates that the disease, in the greater number of cases, is bilateral.

In literature, however, there has always been a great discussion as to whether mb. Menièri was a bilateral or a unilateral ear affection.

Thus Menière[1] (l. c. S. 88) refers to both bi- and unilateral cases, whilst Lucae[5] is of opinion that a bilateral mb. Menièri is most uncommon. Gruber[2] thinks, that the disease is oftenest bilateral, and that in unilateral cases we most likely have to do with hemorrhage of the labyrinth. G. Heermann[1] has published 50 cases, of which 15 are bilateral. L. Baldenweck has 7 cases, of which, at any rate the 4 are bilateral. Thornval[1] publishes 8 cases as unilateral, but of these the one exhibited raised lower limit and lowered upper limit on the sound ear, and 2 lowered upper limit on the sound ear. In the other cases the values are only given as »normal», and but one observation of each patient is quoted.

In a later work Thornval[2] maintains, that mb. Mènieri is almost always unilateral; he considers, that should a bilateral ear affection be present, it would, as far as the other ear is concerned, refer to another disease, for inst. arteriosclerosis or some other condition of hereditary origin. Bárány[3] also believes that this affection is, as a rule, unilateral.

The cause of this great divergence of opinions may, perhaps, be sought in the extremely changeable hearing, and, perhaps, also in the circumstance, that the disease of some periods affects the one ear most, of other periods the other (for inst. Pt. 14, 36); but the principal cause is assuredly, that the pt. is examined at the commencement of the disease, when a proportionally greater number of cases will be found to be unilateral, and when the eventual affection of the so called sound ear is minimal and therefore not considered of any importance.

Investigations of the changing acoustic function. Influence of fluid metabolism.

The more immediate conditions in spontaneous changes in hearing.

Many of our pts have themselves noticed the spontaneously extremely changeable hearing, as found by us.

14 pts (Nos. 2, 3, 12, 14, 18, 34, 51, 52, 53, 54, 59, 90, 91, 126) declare that hearing is worst in the morning, improves during the day and is best in the evening. Some hear best after taking exercise. Thus No. 56 and 82 hear best after running, when they have perspired freely. No. 16 and 50 hear best after long walks; when they begin to feel a pressure on their ears they at once go for walks. No. 16 even gets up in the night in order to find relief from the pressure in the head by walking. No. 13 hears best after having played tennis, no. 3, 14 and 18 after dancing, no. 33 after long bicycle rides.

Some pts (Nos. 3, 14, 24, 41, 51, 132) perspire as a rule with difficulty, and No. 132 has noticed that she hears best and feels best when she perspires, for which reason she often does hard bodily work.

Others have noticed that they hear worse after consumption of liquids, for inst. No. 4 hears worst after meals, No. 13 especially after taking soup. No. 18 hears worst 4—8 hours after drinking soda water or beer; after drinking plain water a longer time elapses before the decrease of hearing sets in.

In other pts the fluctuations of hearing depend upon the weather, for inst. Nos. 14, 16, 38 hear best in warm, No. 82 worst in damp weather, Nos. 2, 27, 37 hear worst when near the sea.

Many have noticed that their buzzings, headache, giddiness, vasomotor rhinitis and diuresis are very changeable, corresponding to their changeable hearing.

Thus 19 pts declare (Nos. 2, 12, 13, 14, 19, 21, 23, 52, 64, 91, 92, 98, 99, 103, 115, 116, 126, 128, 129) that they feel most buzzings and headache in the morning, feel altogether worst then and most easily get their attacks of giddiness.

In several (Nos. 8, 14, 16, 20, 21, 47, 48, 51, 64, 72, 75, 82, 129) the one or the other side of the nose is closed in the morning.

In this connection we may mention, that No. 4 declares hearing to be best after having had a cold in the head.

Some pts, (Nos. 3, 14, 41, 51) have noticed that their diuresis is constantly small (»oliguria»), whilst in others the diuresis varies. Thus Nos. 54 and 85 had copious diuresis in connection with their attacks. No. 114 had an increase of diuresis in connection with her menstruation.

Some (Nos. 14, 43, 54, 56, 66, 78, 107, 125, 131, 132) suffer during the attacks from diarrhoea, others (Nos. 25, 43, 112, 130) from perspiration and epiphora.

In our opinion the above circumstances indicate, that the intake and output of fluid plays a part with regard to both the changing hearing and the attacks of giddiness.

Control of fluid metabolism. Schedules as to the diuresis, weight and intake of fluids of 89 pts have been kept, in order to further investigate the influence of fluid metabolism on hearing. At the same time we made notes as to special perspiration and as to the atmospheric temperature, in order to estimate the presence of a greater extrarenal output. Notes were also made as to obstipation or diarrhoea; but calculation as to the quantity of fluid given off by the skin, lungs and bowel is necessarily but an estimate.

We have reckoned the fluid intake so, that especially watery foods, such as fruit, vegetables and suchlike were either entirely excluded from, or entirely included in the fluid intake. No account was kept of the water content of the solid food, but what each pt consumed was accounted for, and as these pts were all treated in the hospital and their food, therefore, the same for all, and fairly uniform, it has seemed possible to form an estimate as to any particularly great divergences between intake and diuresis, especially as the comparisons made extended over a considerable time and included all the 89 pts. Calculations as to »oxydation water», therefore, are of no importance.

The pts were weighed at least every 5th day, their height was also measured out of consideration to over or under weight.

But few of these daily determinations are given here, on account of the length of the case books.

TABLE I.

Diet with reduced intake of salt and fluid.

Patient No. 15 $^{31}/_{8}$—$^{17}/_{9}$ 26				Patient No. 34 $^{18}/_{1}$—$^{24}/_{2}$ 28				Patient No. 18 $^{3}/_{6}$—$^{27}/_{6}$ 27			
Fluid intake during 24 hours	Diuresis during 24 hours	Difference between intake and output	Hearing of whisper	Fluid intake during 24 hours	Diuresis during 24 hours	Difference between intake and output	Hearing of whisper	Fluid intake during 24 hours	Diuresis during 24 hours	Difference between, intake and output	Hearing of whisper
1500	600	÷ 850	20,00 / 0,40	—	—	—	0,30 / 0,30	—	—	—	2,00 / 1,50
1620	500	÷ 1120	0,10	1750	1100	÷ 650	—	1200	425	÷ 775	—
1250	600	÷ 650	1,00	1600	1000	÷ 600	—	1200	625	÷ 575	—
1450	800	÷ 650	0,25	1750	1150	÷ 600	—	1000	1100	+ 100	2,00 / 0,75
1540	1075	÷ 375	—	2000	1150	÷ 850	—	800	800	0	
1500	1200	÷ 300	—	1800	1000	÷ 800	—	1200	1550	+ 350	
1500	1400	÷ 100	2,00	?	?	?	0,30 / 0,30	500	600	+ 100	2,00 / 2,00
1500	1550	+ 50	2,25	450	750	+ 350	—	500	550	+ 50	4,00 / 6,00
1350	1500	+ 150	2,75	450	900	+ 450	—	500	650	+ 150	2,00 / 4,00
1100	1200	+ 100	3,50	550	550	0	—	500	500	0	4,00 / 10,00
800	750	÷ 50	8,00	650	650	0	—	500	625	+ 125	—

650	800	÷ 150	8,00	600	600	0	—	500	625	+ 125	4,00 / 2,00
700	700	0		500	600	+ 100	—	500	625	+ 125	10,00 / 4,00
1100	1550	+ 350		?	?	—	0,70 / 1,00	500	625	+ 125	—
1100	1300	+ 200	8,00	700	570	+ 50	—	500	600	+ 100	20,00 / 20,00
1100	1900	+ 600	8,00	650	650	0	—	400	500	+ 100	—
1100	1500	+ 400	8,00	730	740	÷ 10	—	350	325	÷ 25	—
—	—	—	—	650	630	÷ 20	—	400	425	+ 25	—
—	—	—	—	500	520	+ 20	—	400	450	+ 50	20,00 / 20,00
—	—	—	—	?		—	0,70 / 1,00	400	500	+ 100	—
—	—	—	—	750	750	0	—	400	500	+ 100	10,00 / 20,00
—	—	—	—	400	450	+ 50	—	400	400	0	—
—	—	Okt. 1926	20,00 / 20,00	600	650	+ 50	—	350	650	+ 300	—
—	—			500	500	0	—	400	475	+ 75	—
—	—	—	—	—	—	—	1,25 / 1,00	350	550	+ 200	—
—	—	—	—	—	—	—	—	350	500	+ 150	20,00 / 20,00

In 31 pts, i. e. ⅓, (Nos. 2, 3, 9, 13, 14, 15, 19, 32, 34, 37, 41, 45, 46, 51, 74, 76, 85, 86, 92, 97, 104, 105, 108, 112, 115, 116, 117, 119, 123, 125, 126) the diuresis was remarkably small in proportion to the intake, for inst. No. 15: intake about 1500, diuresis 500—600, No. 32: intake about 1100, diuresis 600—650, No. 34: intake 1200, diuresis 450—600.

In some few cases the variation in the diuresis was remarkably small, when the intake varied, for inst. No. 3.

Experimental investigations as to the influence of fluid metabolism on hearing. We have endeavoured to influence fluid metabolism experimentally, principally by means of experimentally increased output, in order to examine the effect thus produced on hearing.

Diet with reduced intake of salt and fluid. By this diet alone we obtained in nearly all the pts an increased diuresis and corresponding loss of weight, but as the treatment is a lengthy one, and as it was often necessary to give the pts other treatments such as catheterization of the Eustachian tube, or universal treatment such as massage, gymnastics and baths, at the same time, it is impossible to deny, that these other treatments may have influenced hearing in these cases.

However, the examples on table I seem to show that hearing increases as soon as the diuresis increases, and that this is particularly the case when the diuresis is greater than the fluid intake.

Salyrgan. A quickly acting diuretic, such as salyrgan must, however, be employed when a pure result of increased fluid output is desired.

Salyrgan is a combination of mercury with natriumsalicylamidacetat

There exist numerous investigations as to the action of salyrgan and other diuretics related to it, such as novasurol, regarding which we refer our readers to K n u d M ø l l e r's article on the subject. It is not quite certain whether there is a specific action on the kidneys, as S c h u r has maintained. The principal thing seems to be the mobilisation of fluid in the tissues (N o n n e n b r u c h). At the same time a considerable extra output of Cl Na takes place.

We have made 77 salyrgan experiments on 46 pts with mb. Menièri. Besides this we have tried its effects on a series of other

cases, which we do not quote here; in all we have tried this medicament about 120 times.

It was applied intravenously in doses of 1.0—1.8 ccm in a 10 % solution.

In 2 cases there was a local irritation on account of injection beside the vein. Acute renal trouble appeared in one case, (No. 100) after a 1.5 ccm injection. The pt. weighed 98 kilo, the dose consequently was small in proportion to the weight. Corresponding incidents have been observed in a few other pts not included in this material.

The pts are weighed immediately before the injection, the weight is however wanting in 10 of our first experiments (Exp. 26, 29, 30, 31, 32, 33, 73, 74, 75, 76).

The hearing is tested immediately before the injection and several times again in the course of the ensuing days. The weight, acoustic function, eventual figures for hemoglobin or hematocrit determinations, also length of time after injection when the determination was taken, are all given below.

The diureses are measured per 24 hrs. reckoning from 8 o'clock. On account of space we only give the diuresis and intake for the 24 hrs. previous to, the 24 hrs. of and the 24 hrs. after the injection. The first 24 hours after the injection are marked in italics in the following schedules.

As a rule the injections were given in the forenoon.

In 8 cases (Expm. 39, 45, 46, 67, 68, 73, 74, 75) there was no increase in the diuresis, apparently without any particular cause. All the rest of the patients had distinct increase of diuresis. The increase commences, as a rule, a few hours after the injection, and is especially copious during the first 5 hrs., decreasing afterwards. In some cases the increased diuresis continued for the next 24 hrs. also (for inst. exp. 2, 24, 31, 65, 66). The diuresis often increased to 2—3—4 times the pt's normal diuresis, without any compensatory decrease in the following 24 hrs.

The majority of the pts were on a reduced and, as far as possible, also constant fluid intake with reduced salt, for at least one or two days both before and after the injection. Thus the amount of fluid in the organism was, at all events to a certain extent, already reduced.

On the other hand we gave some few pts a good quantity of fluid before and after the injection, in order also to be able to observe the influence of the more copious intake of fluid (for inst. expm. 15, 31, 36, 45, 47, 58, 66, 68, 73, 74, 75).

Whilst thirst was very often felt in the cases where pt. was put on small intake of fluid, for inst. 500—600 gr., this result was on the opposite — as also the effect on hearing — only exceptionally obtained where the intake of fluid was copious, although constant.

The feeling of thirst was sometimes so troublesome, that 2 pts refused to continue on the reduced fluid intake after the elapse of two days.

Some did not feel thirst in spite of small fluid intake, copious diuresis and corresponding loss of weight.

One pt, expm. 46, increased in weight in spite of copious diuresis, he declared he was thirsty but his tongue was never the less moist, we must therefore suppose that he consumed a greater quantity of fluid than stated. We discovered that No. 6 (not included in above experiments) got up at night and drank from the tap. Both these pts were, for that matter, imbecile. The rest of the pts kept to the cure as far as we could control them by diuresis, thirst and loss of weight.

The loss of weight was often considerable, for inst. No. 100, experiment 57 lost 2,200 grs in the course of 24 hrs. No. 99 in experiment 56 lost 1,300 grs in the course of 7 hrs. No. 126 in experiment 26 lost 2,600 grs in the course of 20 hrs.

The loss of weight was sometimes greater than the corresponding diuresis. In these cases there is then an indubitable increase of extrarenal fluid output. A few of the pts also felt warm and perspired.

The experiments may be arranged in the following groups, according to the rise or nonrise of hearing in conjunction with the injection.

I. Cases with indubitable increase of hearing.

A.	Loss of weight with thirst	28 experiments	(1—28)
B.	» » without »	0 »	
C.	No loss of weight	0 »	
D.	Not weighed (with thirst)	5 »	(29—33)

II. Cases with dubious increase of hearing.

A.	Loss of weight with thirst	5 experiments	(34—38)
B.	» » » without »	6 »	(39—44)
C.	No loss of weight	3 »	(45—47)
D.	Not weighed	0 »	

III. Cases with no improvement of hearing.

A.	Loss of weight with thirst	3 experiments	(48—50)
B.	» » » without »	16 »	(51—66)
C.	No loss of weight	6 »	(67—72)
D.	Not weighed	4 »	(73—76)

IV. Cases with decrease of hearing.
Increase of weight and no thirst 1 experiment (77)

An indisputable improvement in hearing was thus obtained in 33 of 77 experiments.

It must, however, be observed that the pts in 6 of the remaining experiments in group III (Exp. 49, 58, 61, 62, 63, 64) had practically normal hearing prior to the injection. These cases therefore form a separate group, which must be subtracted, when summing up the acoustic result of the effects of salyrgan.

In only 2 cases (Exp. 48, 50) was thirst unaccompanied by increase of hearing (the 3rd case in group III A had a practically normal hearing beforehand). All the remaining cases show, that if an increase of hearing is to be attained, the output of fluid must be so great, that it not only gives loss of weight but also thirst, (group I A).

When the pts are not thirsty in spite of a considerable output of fluid — demonstrable from the increased diuresis and loss of weight (group II B and III B), we may suppose that the body has a sufficient quantity of fluids available, without being obliged to empty the »free fluid» in all the depots, amongst which also such as influence hearing.

We must therefore suppose, that the quantity of salyrgan used in these cases has not been sufficient to evacuate all the depots and thus create thirst, although the diuresis was considerable, often 3—4—5 or indeed 6—8 times as great as in the previous 24 hrs. There must then have been a particularly plentiful reserve of »free fluid».

In some cases it also occured, that thirst and improvement in hearing were obtained first when we repeated the salyrgan experiment and further evacuated fluid, for inst. pt. no. 87 experiment 67 and 19, pt. no. 89 exp. 71 and 28.

Where there is no or only very slight diuretic effect, no loss of weight and of course no thirst, no improvement in hearing appears (Exp. 45, 46, 47, 66, 67).

The rise in hearing may be quite considerable, for inst. exp. 11 from

$$v\,\frac{3{,}00}{1{,}5—2{,}0}—\frac{15{,}00}{15{,}00}\quad \text{exp. 23 } v\,\frac{6{,}00}{aa}—\frac{20{,}00}{0{,}10},\quad \text{exp. 30 } v\,\frac{aa—0{,}25}{0{,}5—4{,}00}—\frac{14{,}00—20{,}00}{10{,}00—20{,}00}$$

This rise of hearing often appears in the course of a few hours, (Exp. 5, 13, 21, 24, 27, 30).

In some cases it seems that the action of the salyrgan is ushered in by a »contra action» on the hearing, there appearing an initial phase with buzzing and diminished hearing. The same was observed in some other salyrgan experiments not included here, in one of these indeed giddiness and nystagmus appeared.

This initial phase, which — as we shall see later on — is more evident when slower acting diuretics are used, may be an indication that simultaneously with an unbinding and evacuation of fluid in kidneys, skin, mucous membranes and bowels, fluid is evacuated temporally also into the ear. There is no thirst during this first phase, often on the contrary salivation. We may in this connection mention, that preliminary investigations as to the volume of the blood — partly by hemoglobin determinations (Sahli-Leitz' apparatus) partly by hematocrit determinations (Allen's apparatus) — indicate, that the volume varies considerably during the effects of the salyrgan, and that both a considerable dilution (Exp. 3, 4, 64, 66, 69) and a considerable concentration can take place (for inst. exp. 6, 11, 17, 18, 21, 28). The latter appeared preeminently in connection with thirst. The above mentioned first phase may, therefore, stand in relation to an initial hydremia or, in any case, to a surplus of »free fluid» in the blood. The changes in the volume of the blood are, however, so irregular and the cases of »contra action» on hearing by salyrgan so few, that we do not deem ourselves

justified for the present in expressing with certainty any opinion as to these problems.

We may here add, that we have observed a similar hydremia in a case treated with salicyl, which treatment is often accompanied by deafness buzzings and giddiness. Hematokrit determinations have been the object of further investigations made by S. F. Nielsen in this department. His observations seem to indicate, that this hydremia is typical to salicyl.

Pt. no. 14 may be given as an instance of the first phase in the action of salyrgan (Expm. 30):

Before the experiment $v\frac{aa—0,25}{0,25—4,00}\ a_1M\frac{0}{0}a_1A\frac{5}{20}Li\ \frac{80}{38}$ 11½ o'clock. Salyrgan 1 ccm.

12 ½ o'clock $v\ \frac{0}{0}\ a_1M\frac{0}{0}a_1A\frac{0}{0}Li\frac{120}{>150}$ pt. herself has the impression of being stone deaf, but can hear the noise of carts in the street. Violent buzzing. 14 o'clock diuresis commences. 15 o'clock increase of hearing and decrease of buzzing and headache.

16 o'clock $v\ \frac{0,20—6,00}{3,00—20,00}\ V\frac{1,0—20,00}{}\ a_1M\frac{vbr.}{vbr.}a_1A\frac{5}{22}Li\frac{50}{38}$

17 ½ o'clock $v\ \frac{1,00—20,00}{20,00}\ a_1M\ \frac{0}{0}\ a_1A\frac{12}{33}Li\frac{45}{36}$

19 ½ o'clock $v\ \frac{0,20—12,00}{20,00}\ a_1M\ \frac{0}{10}\ a_1A\frac{12}{33}Li\frac{38}{30}$

22 o'clock $v\frac{14,00—20,00}{20,00}\ a_1M\ \frac{8}{10}\ a_1A\frac{15}{33}Li\frac{24}{24}$

Violent thirst.

Diuresis on the day of the experiment 1560 against 600 the day before, with an intake of respectively 600 and 800 grs.

Investigations of the acoustic function in salyrgan experiments exhibit the same characteristic features as in the spontaneous rise of hearing, in the relation between lower limit, airconduction, boneconduction and hearing of whisper. They change besides legitimately, in accordance with the weight of the body, in exactly the same way as we have observed in spontaneous changes of hearing.

TABLE II.

Salyrgan experiments.

I. Cases with indubitable increase of hearing.

A. Loss of weight with thirst.

1.

Patient No. 2 7/6 28. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	650	*1250*	550
Specif. weight	1024	*1013*	1024

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	69.7	3.00 / 0	5 / 2	22 / 12	36 / > 150	14000 / 8000	÷	+ / +
24 hrs. later	68.7	7.00 / aa	6 / 5	21 / 18	24 / 65	13500 / 8500	+	÷ / +
31 hrs. later		7.00 / 0.05	7 / 6	21 / 15	26 / 70	14000 / 8000		+ / +
48 hrs. later	68.7	12.00 / 0.05	6 / 6	17 / 14	28 / 70	16000 / 8500	+	

2.

Patient No. 3 28/8 26. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	575	*450*	650
Diuresis » » »	400	*1100*	900
Specif. weight	1028	*1013*	1016

	Weight	v	$a_1M=15$	$a_1A=(60)$	Li	Ls	Thirst	Buzzings
Before Injection	64.0	7.00 / 8.00	17 / 18	47 / 45	20 / 20	13000 / 13000	÷	+
17 hrs later	62.5	15.00 / 20.00	18 / 17	60 / 60	17 / 17	13000 / 13000	+	+
24 hrs. later		20.00 / 20.00	19 / 16	62 / 61	17 / 17		+	

3.

Patient No. 28 9/8 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	200	*650*	200
Specif. weight	1032	*1013*	1032

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	58.0	82.5	20.00 / 2.0—20.00	10 / 12	38 / 24	18 / 36	18000 / 17000	÷	+
21 hrs. later	57.0	73	20.00 / 20.00	13 / 16	48 / 33	15 / 30	17000 / 16000	+	+
30 hrs. later	57.2	73	20.00 / 20.00	12 / 15	44 / 31	16 / 30		÷	÷
46 hrs. later	58	73	20.00 / 2.0—4 00	12 / 11	40 / 26	16 / 34	17000 / 17000	÷	— / +

4.

Patient No. 28 20/2 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 25 hrs.	600	*600*	600
Diuresis » » »	250	*1150*	200
Specif. weight	1034	*1032*	1032

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	54.0	47	10.00 / 0.10	8 / 5	21 / 10	24 / 90	13000 / 12500	÷	+
6 hrs. later	53.5	39.5	10.00 / 0.10	7 / 6	25 / 15	24 / 80	14000 / 14000	+	+
23 hrs. later	53.2	39.5	14.00 / 0.20	11 / 9	29 / 24	17 / 70	14000 / 12000	+	÷
48 hrs. later	53.5	34.5	20.00 / 0.30	11 / 9	30 / 22	17 / 36	14000 / 14000	+	

5.

Patient No. 33 12/9 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	500	*1775*	600
Specif. weight	1025	*1008*	1018

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	74.5	20.00 / 0.75	14 / 10	49 / 25	15 / 36	20000 / 19000	÷	+
2 hrs. later	74	20.00 / 1.50	14 / 14	56 / 25—30	15 / 28	19000 / 18000	+	+
16 hrs. later	72.7	20.00 / 4.00	14 / 15	48 / 28	15 / 20	19000 / 17000	+	+
50 hrs. later	74.0	20.00 / 0.2—1.50	16 / 12	47 / 22	15 / 32	18000 / 17000	(?)	+

6.

Patient No. 34 7/6 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	800	*1200*	500
Diuresis » » »	800	*1550*	600
Specif. weight	1018	*1014*	1025

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	78.6	88	2.00 / 0.75	6 / 10	22 / 36	18 / 18	8000 / 8000	÷	+
6 hrs. later	78	114	2.00 / 2.00	8 / 11	24 / 45	18 / 18	8000 / 9000	÷	+
27 hrs. later	77.7	99	2.00 / 2.00	9 / 10	26 / 41	20 / 18	8500 / 8500	÷	÷
45 hrs. later	77.5	92.4	4.00 / 6.0—10.00	8 / 11	38 / 48	15 / 15	9500 / 9000	+	÷
77 hrs. later	78.2	90.2	2.00 / 4.00	6 / 8	25 / 42	15 / 15	9000 / 8000	÷	÷

7.

Patient No. 43 17/5 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	550	*1700*	375
Specif. weight	1028	*1016*	1026

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	68.7	20.00 / 0.2—0.4	11 / 11	40 / 13	24 / 55	15000 / 16000	÷	+
9 hrs. later	68.2	20.00 / 0.75—1.00	10 / 9	45 / 10	24 / 50	15000 / 16000		
31 hrs. later	68.0	20.00 / 0.2—1.00	9 / 9	45 / 18	19 / 24	20000 / 18000	+	+

8.

Patient No. 43 5/6 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	600
Diuresis » » »	500	*1875*	725
Specif. weight	1031	*1012* —	1024

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	67	81.0	20.00 / 0.75	10 / ?	44 / 25	24 / 36	18000 / 17000	÷	+
5 hrs. later	65.7	82	20.00 / 1.50	15 / ?	48 / 25	18 / 24	19000 / 19000	+	+
27 hrs. later	66.0	88	20.00 / 1.5—2.00	12 / ?	44 / 22	18 / 26	19000 / 18000	+	+

9.

Patient No. 58 29/8 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	*800*	600	
Diuresis » » »	*950*	725	
Specif. weight	*1018*	1024	

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	70.5	103.6	2.00 / 20.00	15 / 20	20 / 42	30 / 15	15000 / 15000	÷	
16 hrs. later	69.2	104.3	10.00 / 20.00	17 / 23	20 / 50	30 / 15	16000 / 15000	+	÷

10.

Patient No. 58 22/9 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	600	*1500*	600
Specif. weight	1030	*1012*	1026

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	66.1	20.00 / 20.00	23 / 23	31 / 51	19 / 15	16000 / 15000	÷	÷
4 ¼ hrs. later	65.2	20.00 / 20.00	18 / 27	44 / 59	28 / 15	15000 / 16000	+	÷
31 hrs. later	65.0	20.00 / 20.00	21 / 27	35 / 58	15 / 15	16000 / 16000	+	÷

11.

Patient No. 60 7/7 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1000	*500*	500
Diuresis » » »		*1300*	375
Specif. weight		*1014*	1027

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst
Before Injection	65.7	67.1	3.00 / 1.5—2.00	9 / 7	13 / 11	22 / 24	11000 / 12000	÷
6 hrs. later	65.2	96.8	2.0—5.0 / 2.00	11 / 9	36 / 17	22 / 18	11000 / 13000	÷
24 hrs. later	64.6	93.5						+
46 hrs. later	64.0	78.1	15.00 / 15.00	11 / 11	37 / 27	19 / 16	9000 / 12000	+

12.

Patient No. 62 22/2 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	375	*1500*	625
Specif. weight	1028	*1008*	1018

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzz.
Before Injection	51.5	40.5	1.50	11	20	24	14000	÷	±
				12	37	17	16000		
27 hrs. later	50	44.5	20.00	9	24	26	14000	+	±
			20.00	11	37	17	15000		
48 hrs. later	50.2	48	6.0—15.00	9	24	26	14000	+	±
			20.00	7	33	15	14000		
72 hrs. later	50.0	47.5	10.00	10	26	28	14000		±
			20.00	11	31	15	15000		
96 hrs. later	49.7	46.0	10.00	12	24	28	14000	+	±
			20.00	12	35	15	15000		

13.

Patient No. 69 5/7 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs. 700 *500* 500

Diuresis » » » 550 *1500* 450

Specif. weight 1028 *1012* 1027

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	68.0	91.3	20.00	11	34	22	14000	÷	+
			0.20—10.00	7	26	24	14000		
8 hrs. later	67.5	96.8	20.00	15	56	17	14000	+	+
			0.40—20.00	10	37	17	14000		
31 hrs. later	67.5	96.8	20.00	14	50	15	15000	+	+
			1.0—20.00	10	35	18	14000		

14.

Patient No. 69 16/8 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs. 500 *400* 600

Diuresis » » » 525 *1250*

Specif. weight 1020 *1013*

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	66	95.9	20.00	14	22	18	14000	÷	+
			0.2—0.4	6	30	24	16000		
6 hrs. later	65	91.6	20.00	14	38	22	15000	÷	+
			0.2—0.4	9	22	28	15000		
23 hrs. later	65	88	20.00	17	47	16	15000	+	+
			1.0—6.0	8	32	24	14000		

15.

Patient No. 72 28/5 1927. Salyrgan 0.8 ccm.

Fluid intake pr. 24 hrs.	1400	*1600*	1200
Diuresis » » »	1000	*1500*	800
Specif. weight	1022	*1014*	1025

	Weight	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	79.5	0 / 0.05	0 / 1.50	0 / 0	0 / 13	>150 / 40	÷ Mon. / 13000	÷	+
22 hrs. later		0 / 0.10	0 / 4.00	0 / 0	0 / 17	150 / 36	÷ Mon. / 14000	÷	+
67 hrs. later	79.0	0 / 0.40	aa / 20.00	0 / 1.2	8 / 20	80 / 22	6000 / 13000	+	+

16.

Patient No. 72 5/6 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	600	*1250*	550
Specif. weight	1034	*1018*	1026

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	78.6	91	0 / 0.10	0 / 0	0 / 8	150 / 36	6000 / 14000	÷	+
5 ½ hrs. later	78.0	97	0 / 0.10	0 / 0	7 / 16	90 / 24	6000 / 14000	+	+
27 hrs. later	77.6	89.1	0 / 0.10	0 / 0	7 / 23	90 / 24	6000 / 14500	+	+
44 hrs. later	77.2	89.1	0 / 0.10	0 / 0	8 / 18	55 / 22	6000 / 14000	+	÷
76 hrs. later	77.5	83.6	0 / 0.10	0 / 0	7 / 19	90 / 24	7000 / 14000	+	+
90 hrs. later	76.7	98.0	0 / 0.40	0 / 0	10 / 25	90 / 24	7000 / 14000	+	+

17.

Patient No. 72 5/7 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	350	*350*	900
Diuresis » » »	450	*1275*	400
Specif. weight	1025	*1011*	1028

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	75.5	94.6	0 / 0.05—0.20	0 / 0	8 / 18	100 / 32	6000 / 13000	÷	+
4 hrs. later		102.3	0 / 0.05—0.20	0 / 6	16 / 20	100 / 28	6000 / 13000	÷	+
8 hrs. later	74.8		0 / 0.05—0.40	vbr. / vbr.	15 / 20	90 / 24	6500 / 13000	+	+
31 hrs. later	74.8	99.9	0 / 0.2—0.75	vbr. / 12. vbr.	13 / 28	65 / 28	6000 / 14000	+	÷

18.

Patient No. 86 20/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1000	*600*	500
Diuresis » » »	400	*1250*	350
Specif. weight	1017	*1009*	1021

	Weight	Hmglb.	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	81.5	85	0.10 / 0	20.00 / 10.00	7 / 0	15 / 17	30 / 36	10000 / 9000	÷	+
22 ½ hrs. later	80.4	105	0.20 / 0.05	20.00 / 15.00	8 / 0	18 / 20	24 / 30	10000 / 9000	+	
30 ½ hrs. later	80.6		0.40 / 0.10		7 / 0	18 / 22	24 / 30	10000 / 9000		

19.

Patient No. 87 24/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	725	*1700*	650
Specif. weight	1028	*1010*	1026

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	88.8	0.30 / 0.40	0 / 0	4 / 8	90 / 45	14000 / 10000	÷	+
5 hrs. later	88.2	0.30 / 0.40	1 / 1	6 / 15	45 / 30	13000 / 10000	÷	+
24 hrs. later	87.7	1.50 / 1.00	0 / 0	12 / 20	28 / 36	13000 / 10000	+	+
27 hrs. later	*87.7*	3.00 / 1.75	0 / 0	12 / 22	36 / 28	14000 / 10000	+	÷

20.

Patient No. 88 11/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	500	*1000*	1375
Specif. weight	1029	*1018*	1009

Only conversation voice determined, as she cannot be brought to understand tuning fork tests.

	Weight	Hmglb.	V
Before	72.6	105	0.05 / 0
24 hrs. later	» 72.4	» 96	» 0.50 / 1.25—20.00

21.

Patient No. 88 12/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	*500*	500	500
Diuresis » » »	1000	*1375*	*500*	625	625
Specif. weight	1018	*1009*	*1010*	1014	1015

	Weight	Hmglb.	v	V	Thirst
Before Injection	72.9	90	0 / 0	0.10 / 075—1.00	
17 hrs. later	71.2	110	aa / aa	20.00 / 20.00	+
41 hrs. later	71.2	100	0.05 / 0.05	20.00 / 20.00	+
72 hrs. later	71.0	80	0.05 / 0.05	10.00—20.00 / 20.00	
12 hrs. later	72.2	90	0 / 0	2.00 / 10.00	+

22.

Patient No. 88 18/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	500	*1350*	625
Specif. weight	1018	*1010*	1015

	Weight	Hmglb.	v	V	Thirst
Before Injection	71.7	93		2.00 / 12.00	
24 hrs. later	70.0	98	0.20 / 0.10	10.0—20.00 / 10.0—20.00	+

23.

Patient No. 115 19/6 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	1250	*1750*	750
Specif. weight	1012	*1010*	1024

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	62.4	6.00 / aa	9 / 0	38 / 17	? / ?	17000 / 17000	÷	— / +
31 hrs. later	62	8.0—12.00 / 0.05	11 / 3	44 / 23	? / ?	17000 / 17000	÷	+
50 hrs. later	62	7.0—10.00 / 0.05	13 / 10	43 / 27	15 / ?	17000 / 17000	+	+
98 hrs. later		20.00 / 0.10	12 / 8	40 / 30	15 / 20	16000 / 16000	+	+

24.

Patient No. 121 27/12 1927. Salyrgan 0.8 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	625	*2500*	900
Specif. weight	1033	*1010*	1024

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	98.5	aa—0.10 / 2.00	2 / 7	14 / 27	55 / 21	13000 / 16000	÷	+
7 hrs. later	97.7	aa—0.10 / 20.00	3 / 8 ½	8 / 28	55 / 15	12000 / 16000	+	÷
72 hrs. later	96.0	aa / 20.00	5 / 9	22 / 35	24 / 16	13000 / 16000	+	÷

25.

Patient No. 122 28/3 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	300
Diuresis » » »	500	*2125*	490
Specif. weight	1019	*1008*	1025

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	68.8	40	20.00 / 0.10—6.00	13 / 9	25 / 15	15 / 24	16000 / 15000	÷	÷
5 hrs. later	67.7	39.5	20.00 / 0.10—6.00	10 / 10	32 / 15	15 / 24	16000 / 15000	÷	÷
23 hrs. later	67.4	38.5	20.00 / 4.0—15.00	15 / 12	35 / 18	15 / 26	16000 / 15000	÷	÷
96 hrs. later	67.0		20.00 / 20.00	15 / 15	40 / 30	15 / 20	16000 / 16000	+	÷

26.

Patient No. 126 10/7 1928. Salyrgan 1.5.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	650	*2250*	625
Specif. weight	1027	*1010*	1021

	Weight	v	$a_1M=15$	$A_1a=60$	Li	Ls	Thirst	Buzzings
Before Injection	67.6	20.00 / 0.10— 0.50	16 / 15	58 / 16	15 / 20	16000 / 13000	÷	— / +
20 hrs. later	65.0	20.00 / 2.0 —20.00	16 / 18	52 / 31	15 / 21	16000 / 13000	+	— / +
44 hrs. later	65.0	20.00 / 2.0 —20.00	19 / 21	52 / 37	15 / 23	16000 / 12000	+	— / +

27.

Patient No. 134 4/4 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	1400	*600*	600
Diuresis » » »	1000	*2250*	625
Specif. weight	1017	*1008*	1012

	Weight	v	$A_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	95.7	2.50 / 0.30—1.25	10 / 5	25 / 22	24 / 28	12000 / ÷ Mon.		+
7 hrs. later	95	20.00 / 0.30—4.00	11 / 9	35 / 27	15 / 20	12000 / ÷ Mon.	+	+
25 hrs. later	94.2	20.00 / 1.25—6.00	11 / 10	37 / 27	15 / 21	14000 / ÷ Mon.	+	

28.

Patient No. 89 21/11 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	875	*1875*	875
Specif. weight	1020	*1014*	1020

	Weight	Hmglb.	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	61.0	103.4	0.30— 4.00 / 20.00	13 / 13	28 / 50	23 / 18	17000 / 17000		+ / —
4 hrs. later		114.4	0.30— 0.50	13 / 13	28 / 49	23 / 18			+ / — / —
24 hrs. later	60.5	104.5	1.0 — 6.00 / 20.00	17 / 19	32 / 53	23 / 18	18000 / 19000	+	+ / —
72 hrs. later	60.2		2.0 —10.00 / 20.00	14 / 21	32 / 53	18 / 18	16000 / 16000	+	+ / —

D. Not weighed.

29.

Patient No. 3 5/10 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	950	*750*	750
Diuresis » » »	700	*1050*	650
Specif. weight	1023	*1022*	1018

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	1.25 / 0.75	15 / 16	53 / 45	26 / 32	13000 / 13000	÷	+
24 hrs. later	3.00 / 2.50	16 / 14	45 / 43	30 / 34	13000 / 13000	+	+
72 hrs. later	4.00 / 4.00	16 / 18	56 / 50	26 / 24	13000 / 13000		+

30.

Patient No. 14 28/8 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	800	*600*	600
Diuresis » » »	600	*1560*	
Specif. weight	1030	*1013*	

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	aa — 0.25 / 0.5 — 4.00	0 / 0	5 / 20	80 / 38	14000 / 14000	÷	+
8 hrs. later	0.2 —12.00 / 20.00	0 / 10	12 / 33	38 / 30	14000 / 14000	+	+
11 hrs. later	14.00—20.00 / 20.00	8 / 10	15 / 33	24 / 24	14000 / 14000	+	÷
22 hrs. later	14.00—20.00 / 10.00—20.00	0 / 0	12 / 30	40 / 36	14000 / 14000		+

31.

Patient No. 14 13/10 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1100	*1250*	2500
Diuresis » » »	900	*1350*	2500
Specif. weight	1018	*1010*	1005

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	0.10 / aa	0 / 0	25 / 8	38 / 36	11000 / 10000		+
3 hrs. later	0.20 / 2.00	0 / 0	15 / 8			+	+
6 hrs. later	0.20 / 1.00	0 / 0	30 / 15	36 / 45	11000 / 11000	+	+
22 hrs. later	0.20 / 2.00	0 / 0	20 / 10	60 / 60	11000 / 11000		

Attack of giddines one hour after injection; during beginning of attack nystagmus, and again afterwards.

32.

Patient No. 36 17/9 1927. Salyrgan 1.0 ccm.
Much perspiration; fluid intake c. 750, diuresis c. 825 (estimated).

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	0.40 / 20.00	3 / 7	6 / 55	90 / 15			+
48 hrs. later	0.80 / 20.00	6 / 6	14 / 55	55 / 15		+	+

33.

Patient No. 66 27/9 1926. Salyrgan 1.0 ccm.
Out patient. Diuresis estimated as unaltered.

	v	$a_1M=15$	$a_1A=60$	Li	Thirst
Before Injection	3.00 / 20.00	20 / 20	16 / 60	70 / 18	
18 hrs. later	3.50 / 20.00	20 / 14	18 / 65	36 / 15	+
72 hrs. later	8.00 / 20.00	18 / —	14 / 56	32 / 15	+

II. Cases with dubious increase of hearing.

A. Loss of weight with thirst.

34.

Patient No. 90 31/8 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	725	*1350*	625
Specif. weight	1027	*1018*	1026

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	84	20.00 / 0.10—1.50	13 / 8	36 / 15	16 / 80	16000 / 15000	÷	
16 hrs. later	83.7	20.00 / 0.10—0.50	12 / 5	37 / 15	16 / 80	16000 / 14000	÷	— / +
19 ½ hrs. later	83.5	20.00 / 0.4—2.00	14 / 9	40 / 15	16 / 65	16000 / 15000	+	— / +

35.

Patient No. 98 23/5 1928. Salyrgan 1.7 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	375	*2125*	425
Specif. weight	1032	*1010*	1029

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	74.2	20.00 / 0.10	10 / 5	29 / 12	20 / 90	16000 / 13000	÷	÷
24 hrs. later	72.4	20.00 / 0.10	9 / 5	25 / 12	20 / 120	16000 / 13000	÷	÷
46 hrs. later	72.3	20.00 / 0.30	10 / 8	29 / 16	20 / 90	17000 / 13000	+	÷
53 hrs. later		20.00 / 0.40	10 / 8	29 / 16	20 / 36		+	÷

36.

Patient No. 105 24/2 1928. Salyrgan 1.8 ccm.

Fluid intake pr. 24 hrs.	1300	*1300*	1700	1500
Diuresis » » »	750	*2850*	625	875
Specif. weight	1032	*1015*	1025	1026

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	80.0	49	20.00 / 0.05—0.10	8 / 7	27 / 17	15 / 24	12000 / 9500	÷	÷
4 hrs. later	78.5	46 ½	— / 0.20	10 / 9	28 / 17	15 / 24			÷
22 hrs. later	78.0	46	— / 0.20	9 / 8	31 / 17	15 / 26	12000 / 9500	+	÷
72 hrs. later	78.5	40	— / 0.10	9 / 5	27 / 17	15 / 26	12500 / 10000	+	÷
120 hrs. later	77.7	40	— / 0.75	7 / 8	27 / 18	15 / 24	13000 / 10000	+	

37.

Patient No. 109 22/6 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	375	*1375+*	250
Specif. weight	1025	*1010*	1028

	Weight	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	67.0	20.00 / 0	Bá / < aa	9 / 8	31 / 10	20 / > 150	11000 / 11000		+
24 hrs. later	65	—	— / aa	9 / 9	37 / 12	20 / 36	15000 / 12000	+	+

38.

Patient No. 109 4/7 1928. Salyrgan 0.8 ccm.

Fluid intake pr. 24 hrs.	400	*400*	600
Diuresis » » »	250	*1950*	350
Specif. weight	1027	*1010*	1020

	Weight	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	65.7	< 20.00 / 0	— / 0	7 / 7	29 / 17	65 / 65	13000 / 11000	÷	+
18 hrs. later	64.0	20.00 / 0	Bá / 0.10	7 / 6	33 / 16	24 / 36	16000 / 13000	+	— / +

B. Loss of weight without thirst.

39.

Patient No. 106 12/3 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1400	*600*	500
Diuresis » » »	1400	*1000*	875
Specif. weight	1026	*1020*	1022

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	83.0	48.5	20.00 / 0.10	6 / 6	27 / 6	15 / 30	15000 / 14000	÷	+
7 hrs. later	82.5	45	0.10—1.50	9 / 4	26 / 6	18 / 24	16000 / 14000	÷	+
26 hrs. later	81.5	48	0.10—1.00	9 / 6	23 / 7	18 / 26	15000 / 13000	÷	— / +

40.

Patient No. 98 1/6 1928. Salyrgan 1.3 ccm.

Fluid intake pr. 24 hrs.	500	*400*	500
Diuresis » » »	350	*2300*	350
Specif. weight	1028	*1006*	1025

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	74.9	— 0.10	8 5	22 7	20 150	16000 13000	÷	÷
7 hrs. later	73.9	— 0.20	8 5	— 11	20 90		÷	÷
24 hrs. later	73	— 0.20	9 5	25 8	20 90		÷	÷

41.

Patient No. 98 10/5 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	550	*550*	550
Diuresis » » »	500	*1900*	250
Specif. weight	1018	*1010*	1030

	Weight	v	V	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	76.3	— 0	Bá 0.30	9 5	30 8	22 > 150	17000 14000	÷	÷
21 hrs. later	74.7	20.00 0.05	—	10 5	32 7	19 > 150	18000 18000	÷	÷
47 hrs. later	75.2	— 0.20		8 5	— 14	— 130	— 13000	÷	÷

42.

Patient No. 109 16/7 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	400	*400*	400
Diuresis » » »	350	*1650*	400
Specif. weight	1023	*1008*	1020

	Weight	v	V	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	64.3	10.0—15.00 0	0.10	8 7	26 12	24 36	16000 13000	÷	— +
27 hrs. later	63.3	15.00 0	— 0.30	7 6	36 18	24 24	14000 12000	÷	— +
49 hrs. later	63.4		— 0.30	7 6	34 18	24 24	14000 12000	÷	

43.

Patient No. 134 19/4 1928. Salyrgan 1.2 ccm.

Fluid intake pr. 24 hrs.	1000	*1000*	1000
Diuresis » » »	600	*2125*	700
Specif. weight	1017	*1014*	1016

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	92.5	20.00 / 0.20	15 / 7	34 / 14	15 / 45	13000 / 7000	÷	÷
24 hrs. later	91.5	— / 0.20	15 / 7	29 / 17	15 / 24	13500 / 6500	÷	÷
52 hrs. later	91.9	— / 0.20	15 / 10	— / 20	15 / 24	13000 / 6000	÷	÷

44.

Patient No. 122 9/3 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	500	*1500*	500
Specif. weight	1033	*1012*	1022

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	70	38 ½	20.00 / 0.10	8 / 9	25 / 7	15 / 28	14000 / 14000	÷	÷
2 hrs. later		40 ½	— / 0.10	7 / 6 ½	27 / 7	15 / 30	14000 / 14000	÷	÷
8 hrs. later	69.0	40 ½	— / 0.10	8 / 9	25 / 13	15 / 28	15000 / 14000	÷	÷
27 hrs. later	68.5	40 ½	— / 0.10—1.00	8 ½ / 8	26 / 12	15 / 28	15000 / 15000	÷	÷
32 hrs. later	69.3	38 ½	— / 0.10—1.50	9 / 9	29 / 12	15 / 26	15000 / 15000	÷	÷
74 hrs. later	69.5	35 ½	— / 0.20—1.50	8 ½ / 8	40 / 16	15 / 23	15000 / 15000	÷	÷

C. No loss of weight.

45.

Patient No. 29 15/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1250	*1350*	1250
Diuresis » » »	1015	*1375*	725
Specif. weight	1022	*1020*	1024

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	60	0.40	15/10	52/33	15/36	13000/13000	÷	—
5 hrs. later		0.50	13/12	55/33	15/26	13000/13000	÷	—/+
22 hrs. later	60	0.50	17/12	68/35	15/24	12500/12500	÷	—/+
29 hrs. later	60	0.75	16/15	70/45	15/23	13500/13000	÷	—/+

46.

Patient No. 25 28/6 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1400	*1100*	500
Diuresis » » »	1250	*1150*	550
Specif. weight	1010	*1012*	1024

	Weight	Hmglb.	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	73.9	101.2	aa/aa	0.10—0.75/0.20—1.00	10/7	0/18	>150/150	10000/9000	÷	+
4 ½ hrs. later	74.4	106.7	aa—0.30/aa		11/11	12/17	90/90	11000/10000	÷	+
24 hrs. later		107.8								
28 hrs. later			aa/aa	0.10—0.75/0.20—0.40	10/10	4/18	90/90	12000/11000	÷	+
72 hrs. later	74.0		aa/aa	0.10—3.00/0.10—3.00	15/12	8/22	40/80	12000/10000	÷	

47.

Patient No. 39 24/2 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	1300	*1500*	1550
Diuresis » » »	950	*2000*	850
Specif. weight	1015	*1012*	1015

	Weight	Hematokrit	V	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	67.5	41.5	0.25 / 0.25—0.35	0 / 0	7 / 7	>150 / >150	÷ M / 6000	÷	+
4 hrs. later	67.0	43.0	0.15 / 0.40					÷	+
24 hrs. later	67.7	40.5	0.15 / 1.0 —2.00	0 / 0	0 / 12	>150 / 90	11000 / ÷ M	÷	+
72 hrs. later	68.2	34.0	0.15 / 1.0 —2.00	0 / 0	5 / 13	>150 / 90	÷ M / 12000	÷	+

III. Cases with no improvement of hearing.

A. Loss of weight with thirst.

48.

Patient No. 86 23/10 1927. Salyrgan 1.2 ccm.

Fluid intake pr. 24 hrs.	500	*600*	500
Diuresis » » »	300	*2100*	400
Specif. weight	1022	*1008*	1017

	Weight	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	78.8	0.80 / 0.20	20.00 / 20.00	5 / 1	16 / 16	26 / 30	9000 / 9000	÷	
4 hrs. later	78.7	0.30 / 0.20		2 / 0	17 / 16	24 / 30	10000 / 11000		+
24 hrs. later	77.8	0.80 / 0.20		6 / 0	32 / 24	24 / 24	12000 / 11000	+	+
45 hrs. later	77.5	0.80 / 0.20		6 / 5	17 / 25	24 / 26	14000 / 11000	+	+

49.

Patient No. 97 20/4 1928. Salyrgan 1.3 ccm.

Fluid intake pr. 24 hrs.	500	*600*	600
Diuresis » » »	750	*1250*	625
Specif. weight	1030	*1018*	1030

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	88.7	45½	20.00 / 20.00	14 / 15	35 / 40	15 / 15	16000 / 16000	÷	÷
6 hrs. later	88.2	45	20.00 / 20.00	11 / 15	34 / 40	15 / 15		+	÷

50.

Patient No. 105 5/3 1928. Salyrgan 1.8 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	750	*2750*	875
Specif. weight	1030	*1010*	1020

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	78.2	45	0.10—1.50	11 / 9	29 / 17	15 / 24	12000 / 10000	÷	÷
½ hrs. later		43	0.10—1.50	15 / 13	30 / 19	15 / 23	13000 / 10000	÷	
3 hrs. later		45	0.10—1.00	10 / 10	36 / 19	15 / 26	12000 / 10000	÷	÷
8 hrs. later		45	0.10—0.50	11½ / 10	30 / 19	15 / 26	12000 / 10000	÷	÷
27 hrs. later	77.2	45	0.10—1.00	10 / 9	29 / 19	15 / 24	13000 / 10000	+	÷
51 hrs. later	76.8	45	0.10—3.00	11 / 10	27 / 17	15 / 24	12000 / 10000	+	÷

B. Loss of weight without thirst.

51.

Patient No. 115 10/7 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	825	*1750*	875
Specif. weight	1023	*1010*	1020

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	62.0	20.00 / 0.10—0.50	16 / 10	47 / 20	15 / 23	16000 / 16000	÷	+
22 hrs. later	60.5	/ 0.10	9 / 7	46 / 25	15 / 23	16000 / 16000	÷	— / +
46 hrs. later	60.4	/ 0.20—0.30	13 / 11	55 / 32	15 / 21	16000 / 13000		— / +

52.

Patient No. 33 20/6 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »		*1750*	500
Specif. weight		*1010*	1018

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	69	20.00 / 0.50—4.00	15 / 10	50 / 25	15 / 23	19000 / 19000	÷	— / +
26 hrs. later	67.7	/ 0.50—8.00	19 / 11	50 / 25	15 / 24	19000 / 19000	÷	— / +
49 hrs. later	68.3	/ 0.50—8.00	19 / 12	44 / 25	15 / 26			— / +

53.

Patient No. 33 4/7 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	450	*450*	450
Diuresis » » »	650	*2000*	650
Specif. weight	1031	*1011*	1028

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	69.5	20.00 / 0.50—10.00	12 / 11	55 / 25	15 / 24	19000 / 19000	÷	— / +
18 hrs. later	67.5	20.00 / 2.0—6.00	12 / 11	55 / 25	15 / 24		÷	— / +
42 hrs. later	68.0	20.00 / 2.0—6.00	12 / 11	55 / 25	15 / 24		÷	— / +

54.

Patient No. 54 6/3 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*535*	600
Diuresis » » »	475	*2350*	400
Specif. weight	1028	*1010*	1030

	Weight	Hematokrit	v	$a_iM=15$	$a_iA=40$	Li	Ls	Thirst	Buzzings
Before Injection	75.9	38.5	20.00 / 20.00	7 / 10	19 / 27	22 / 15	16000 / 16000	÷	+ / —
4 hrs. later	74.0	39.5	10.00 / 20.00	7 / 12	17 / 24	24 / 15	16000 / 16000	÷	+
20 hrs. later	73.5	40.5	0.50—1.50 / 20.00	6 / 9	20 / 30	24 / 15	15000 / 16000	÷	+ / — / ÷
28 ½ hrs. later		40	3.00 / 20.00	7 / 12	20 / 32	24 / 15	15000 / 16000	÷	+ / — / ÷
45 hrs. later	73.5	41.5	8.0—20.00 / 20.00	9 / 12	23 / 30	15 / 15	16000 / 17000	÷	÷ / — / ÷

55.

Patient No. 99 7/5 1928. Salyrgan 1.3 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	675	*2000*	625
Specif. weight	1030	*1020*	1028

	Weight	v	$a_iM=15$	$a_iA=40$	Li	Ls	Thirst	Buzzings
Before Injection	89.0	20.00 / aa	10 / 8	24 / 15	18 / 60	14000 / 13000	+	— / +
7 hrs. later	87.7	20.00 / 0.05	7 / 6	26 / 11	18 / 90—100	14000 / 12000	÷	— / +
24 hrs. later	87.5	— / 0.10	7 / 6	29 / 14	18 / 90	12000 / 11500	÷	— / +
48 hrs. later	88	— / 0.20	10 / 8	29 / 22	18 / 65	13000 / 12000	÷	+

56.

Patient No. 99 10/5 1928. Salyrgan 1.2 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	550	*2000*	625
Specif. weight	1030	*1020*	1028

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	87.9	0.20	9/7	26/16	18/90	14000/11000	÷	+
24 hrs. later	86.9	0.20	9/8	37/14	15/80	14000/11000	÷	÷
48 hrs. later	86.9	0.20	7 ½/7 ½	31/16	15/80	13000/11000	÷	÷

57.

Patient No. 100 11/4 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	375	*2850*	300
Specif. weight	1025	*1010*	1020

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	100.2	44.5	10.0—15.00/20.00	8/8	24/35	24/18	16000/16000	÷	+
23 hrs. later	98.0	47	20.00/20.00	9/11	24/34	24/18	16000/16000	÷	÷
54 hrs. later		48.5							

58.

Patient No. 104 27/2 192 . Salyrgan 1.9 ccm.

Fluid intake pr. 24 hrs.	1100	*1100*	1100
Diuresis » » »	650	*2200*	550
Specif. weight	1032	*1010*	1033

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	84.2	45.5	20.00/20.00	11/12	27/27	15/15	17000/17000	÷	+
23 hrs. later	82.5	45	20.00/20.00	10/10	27/31	15/15	17000/17000	÷	÷
47 hrs. later	83.5	47.5	20.00/20.00	9/9	25/27	15/15	17000/17000		

59.

Patient No. 109 6/6 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	*600*	500
Diuresis » » »	*1500*	260
Specif. weight	*1010*	1022

	Weight	v	V	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	67.2	20.00 / 0	0	8 / 0	27 / 0	28 / > 150	12000 / — M	÷	+ / —
23 hrs. later	66.2	20.00 / 0	aa?	7 / 0	28 / 0	24 / > 150	14000 / — M	÷	

60.

Patient No. 111 1/6 1928. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	375	*1800*	400
Specif. weight	1030	*1014*	1022

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	60.9	0.75 / 20.00	12 / 15	12 / 38	55 / 17	16000 / 17000	÷	÷
7 hrs. later		1.00	14 / 15	15	45		÷	÷
24 hrs. later	60,0	1.00	14 / 15	12 / 28	45		÷	÷

61.

Patient No. 127 9/3 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	650	*600*	600
Diuresis » » »	400	*1125*	375
Specif. weight	1029	*1015*	1030

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	59.3	36.5	20.00 / 20.00	16 / 16	32 / 22	15 / 55	15000 / 16000	÷	
2 hrs. later		44	20.00 / 20.00	16 / 16	30 / 22	55		÷	÷

	Weight	Hematokrit	$a_1M=15$	$a_1A=40$	Li	Thirst	Buzzings
8 hrs. later	59.0	47	17/16	35/22	15/38	÷	÷
27 hrs. later	58.5	50	17/17	33/22	15/40	÷	÷

62.

Patient No. 127 26/3 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	375	*1400*	375
Specif. weight	1035	*1013*	1097

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	59.0	41.5	20.00/20.00	22/22	45/32	15/15	17000/17000	÷	÷
6 hrs. later	58.5	45	20.00/20.00	22/22	45/35	15/15		÷	÷
23 hrs. later	58.2	45.5	20.00/20.00	22/22	43/34	15/15	16500/17000	÷	÷
48 hrs. later	58.0	41.0	20.00/20.00	22/22	41/40	15/15	16500/17000	÷	÷

63.

Patient No. 128 26/3 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	250	*1500*	300
Specif. weight	1034	*1015*	1026

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	75.4	47	20.00/20.00	12/10	44/32	15/15	15000/15000	÷	÷
6 hrs. later	74.5	45.5	20.00/20.00	11/9	35/31	15/18	16000/15000	÷	÷
23 hrs. later	74.5	44.5	20.00/20.00	11/10	36/36	15/16	17000/15000	÷	÷
48 hrs. later	74.3	44	20.00/20.00	10/10	34/33	15/15	16000/15000	÷	÷

64.

Patient No. 128 10/4 1928. Salyrgan 1.00 ccm.

Fluid intake pr. 24 hrs.	400	*400*	400
Diuresis » » »	310	*2050*	375
Specif. weight	1032	*1014*	1026

	Weight	Hematokrit	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	74	50.5	20.00 / 20.00	10 / 11	34 / 35	15 / 15	16000 / 15000	÷	÷
6 hrs. later		43.5							
24 hrs. later	72.2	45.5	20.00 / 20.00	12 / 12	34 / 34	15 / 15		÷	÷
72 hrs. later		44	20.00 / 20.00	12 / 15	37 / 36	15 / 15	16000 / 15000	÷	÷

65.

Patient No. 130 27/6 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	500	*500*	500
Diuresis » » »	850	*2000*	1100
Specif. weight	1022	*1009*	1017

	Weight	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	B zzings
Before Injection	83.0	0 / 20.00	0 / —	10 / 12	0 / 40	>150 / 15	÷ M / 9000	÷	÷
24 hrs. later	80.8	0 / —	0 / —	6 / 9	0 / 37	— / 15	÷ M / 10000	÷	÷
48 hrs. later	80.8	0 / —	0 / —	7 / 8	0 / 50	— / 15	÷ M / 12000	÷	÷

66.

Patient No. 12 14/8 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1200	*1200*	1200
Diuresis » » »	950	*1550*	1350
Specif. weight	1017	*1013*	1010

	Weight	Hmglb	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	58.7	102.5	20.00 / 0.5—6.00	8 / 11	45 / 20	15 / 45	17000 / 15000	÷	— / +
2 hrs. later		91.6							— / +
6 hrs. later	57.9		20.00 / 0.2—6.0	14 / 0	45 / 21	16 / 45	18000 / 16000	÷	— / +
8 hrs. later	58.2	82.8	20.00 / 1.5—6.00	15 / 6	45 / 15	16 / 45		÷	— / +
23 hrs. later	58.2	76.3	20.00 / 0.2—6.00	13 / 5	50 / 14	16 / 60	18000 / 18000	÷	— / +

C. No loss of weight.

67.

Patient No. 87 21/10. Salyrgan < 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	825	*950*	600
Specif. weight	1025	*1022*	1030

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	88.9	0.20 / 0.35	0 / 0	4 / 15	90 / 45	12000 / 10000	÷	+
24 hrs. later	88.6	0.30 / 0.40	0 / 0	3 / 15	55 / 36	14000 / 10000	÷	+
72 hrs. later	88.8	0.30 / 0.40	0 / 0	4 / 8	90 / 45	14000 / 10000	÷	+

68.

Patient No. 29 22/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1400	*1400*	1400
Diuresis » » »	875	*1375*	625
Specif. weight	1026	*1020*	1028

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	60	20.00 / 0.30	28 / 16	78 / 33	15 / 24	14000 / 14000	÷	— / +
7 hrs. later	60	— / 1.50	21 / 18	— / 36	15 / 26	— / 14000	÷	— / +
24 hrs. later	60	— / 1.00	21 / 15	— / 36	15 / 24	— / 14000	÷	— / +

69.

Patient No. 75 7/7 1927. Salyrgan 1.0 ccm.

Fluid intake 400 gr. Diuresis 450 gr during first 17 hrs.

	Weight	Hmgbl	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	81.2	110	20.00 / 20.00	12 / 13	40 / 49	28 / 26	15000 / 17000	÷	+
½ hr. later		97.9							
1 hr. later		92.4	20.00 / 20.00	12 / 10	32 / 46	28 / 24	15000 / 17000	+	+
17 hrs. later	81.4	96.8							
24 hrs. later	81.2	96.8	20.00 / 20.00	10 / 10	45 / 48	30 / 30	15000 / 17000	÷	+

70.

Patient No. 33 22/9 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	700	*500*	500
Diuresis » » »	500	*1500*	550
Specif. weight	1026	*1012*	1026

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	73.7	20.00 / 10.0—20.00	20 / 15	46 / 15	15 / 24	19000 / 19000	÷	— / +
3 ¼ hrs. later	74.0	20.00 / 0.2—20.00	18 / 18	60 / c.20	15 / 24	20000 / 19000	÷	— / ++
31 hrs. later	74.0	20.00 / 0.2—20.00	22 / 13	60 / 18	15 / 24	19000 / 19000	÷	— / +

71.

Patient No. 89 26/10 1927. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	625	*1300*	625
Specif. weight	1019	*1025*	1025

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	61.7	0.10 / 20.00	15 / 17	21 / 45	30 / 24	18000 / 18000	÷	+ / —
8 hrs. later	62.4	0.10 / 20.00	11 / 12	21 / 48	36 / 24	17000 / 18000	÷	+ / —
24 hrs. later	61.6	0.10 / 20.00	15 / 15 ½	24 / 49	28 / 23	18000 / 18000	÷	+ / —
48 hrs. later	61.5	0.20 / 20.00	15 / 15 ½	25 / 53	24 / 15	18000 / 19000	÷	—

72.

Patient No. 3 1/9 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	2485	*2175*	900
Diuresis » » »	900	*1250*	400
Specif. weight	1015	*1013*	1028

	Weight	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	65.5	1.00 / 1.25	15 / 16	51 / 50	28 / 28	13920 / 13920	÷	+
4 hrs. later		1.00 / 1.25	15 / 13	42 / 44	28 / 28	13920 / 13920	÷	+
24 hrs. later	65.5	1.00 / 0.75	14 / 16	48 / 44	24 / 24	13920 / 13920	÷	+

D. Not weighed.

73.

Patient No. 13 10/9 1926. Salyrgan 0.8 ccm.

Fluid intake pr. 24 hrs.	950	*1100*	1100
Diuresis » » »	900	*950*	1100
Specif. weight	1023	*1023*	1014

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	1.25	12	34	28	15000	÷	+
24 hrs. later	1.25	10	18	28		÷	+
72 hrs. later	0.75	8	32	30		÷	+

74.

Patient No. 23 11/11 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1500	*1500*	1500
Diuresis » » »	1125	*1125*	1125
Specif. weight	1016	*1026*	1022

	v	V	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	0.10 / 0.10	8.00 / 5.00	6 / 6	25 / 22	38 / 30	13000 / 13000	÷	+
4 hrs. later	0.10 / 0.10	—	5 / 5	22 / 20	36 / 32	13000 / 13000	÷	+
24 hrs. later	0.10 / 0.10	8.00 / 5.00	4 / 4	27 / 22	32 / 32	13000 / 12000	÷	+

75.

Patient No. 55 2/9 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	1100	*1100*	1100
Diuresis » » »	500 +	*900*	1150
Specif. weight	1018	*1012*	1015

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzings
Before Injection	20.00 / 0	11 / 13	58 / 8	23 / > 150	13000 / 7000	÷	— / +
23 hrs. later	— / aa	11 / 13	54 / 10	23 / > 150	13000 / 7000	÷	— / +

76.

Patient No. 13 20/9 1926. Salyrgan 1.0 ccm.

Fluid intake pr. 24 hrs.	950	*950*	
Diuresis » » »	700	*1100*	
Specif. weight	1025	*1020*	

	v	$a_1M=15$	$a_1A=60$	Li	Ls	Thirst	Buzzing	Perspiring
Before Injection	20.00 / 2.00	17 / 12 ½	65 / 36	15 / 28	15000 / 15000	÷	+	÷
24 hrs. later	20.00 / 4.00	18 / 11	65 / 35	18 / 28	15000 / 15000	+	+	+

IV. Cases with decrease of hearing.

77.

Patient No. 2 15/6 1928. Salyrgan 1.5 ccm.

Fluid intake pr. 24 hrs.	600	*600*	600
Diuresis » » »	500	*2500*	475
Specif. weight	1024	*1007*	1021

	Weight	v	$a_1M=15$	$a_1A=40$	Li	Ls	Thirst	Buzzings
Before Injection	68	0.50—1.00 / 0.05	0 / 0	8 / 12	40 / > 150	15000 / 7500		+
15 hrs. later	66.2	0.50 / 0.05	0 / 0	7 / 12	38 / 90	11000 / 8500	÷	+
90 hrs. later	69.0	0.40 / aa	0 / 0	9 / 11	90 / > 150	11000 / 8500	÷	+

The changes in hearing are not proportionate to the increase in the diuresis, but to the body's total loss of fluid (weight), and are intimately associated with the appearence of thirst. There seems then to be no question of any specific action of salyrgan on the organ of hearing.

Diuretics of the purin group. Of these we have tried *agurin* = theobrominnatrium — natriumacetate (Nos. 13, 14, 16, 29, 36, 37, 41, 53, 64, 66, 79) and *diuretin* = theobrominnatrium — natriumsalicylate (Nos. 3, 14, 24, 54, 61), also *euphyllin* = theophyllin — æthylendiamid (Nos. 7, 8, 14, 20, 32, 43, 51, 71, 76).

We will not enter more particularly into the manner in which these drugs act, but simply state that Johannes Bock, by whose advise we have tried them, distinctly points out, that a certain presence of fluid in the organism is requisite for a copious diuresis, and he adds, that the action depends to a great degree upon the influence of the vasomotors. This is, perhaps, the reason why these medicaments have had no diuretic effect on several of our pts. But the hearing has always improved in all the cases where an increased diuresis was obtained. As these medicaments are of slower action than salyrgan, the causal conjunction is, however, not so convincing. When these medicaments are used we find, almost more plainly than with salyrgan, a first phase with bad hearing, increased buzzing and salivation, followed by a phase with increasing hearing and thirst.

As an instance may be given:

Pt. no. 14 before experiment $v\,\frac{10{,}00}{10{,}00}\ a_1M\,\frac{9}{4}\,a_1A\,\frac{22}{38}\,Li\,\frac{24}{24}\,Ls\,\frac{14000}{14000}$

30 ctgrs of euphyllin were given. 5 hrs. later diuresis 500, 7 hrs. later 200 more, in the course of 12 hrs. a diuresis of 1000 grs without pt. having drunk anything in the course of the 12 hrs. The buzzing which had been quite slight before euphyllin was given, increased so much in 2 hrs. as to be like the sound of a waterfall; it remained constant for 12—14 hrs. and then decreased. The hearing decreased as buzzing increased, so much so that pt. was unable to hear conversation. 3 hrs. after euphyllin was taken we found $a_1M\,\frac{0}{0}\,a_1A\,\frac{5}{5}\,Li\,\frac{90}{> 150}\,Ls\,\frac{15000}{14000}$ After this hearing increased in the course of the night, but was not measured until 24 hrs. later and was then $v\,\frac{10{,}00}{10{,}00}$

In nos. 7 and 20 a considerable improvement in hearing took place 2 days after euphyllin treatment was begun, pts at the same time feeling perfectly well and free from giddiness and headache. In a series of other cases, where there was improvement of hearing, the action of the diuretic is not absolutely convincing, as other treatment also was employed.

In a few cases (Nos. 9, 36) we have also made use of calcium-diuretin, which was recently recommended by Berberich, who has published good results obtained with it »bei der sogenannte Altersschwerhörigkeit».

All the diuretics used act then in the same way, the increased hearing being proportional to thirst and loss of weight.

Increased intake of salt productive of thirst. As thirst would seem to be necessary for an increase of hearing, it seemed obvious that we should endeavour to produce this feeling by giving our pts. great quantities of salt. This was tried in some cases. Hearing did also increase after an intake of 20 gr. of salt. But as these experiments are disagreable, not only on account of the thirst, but also on account of the nausea they give rise to, we have not been able to continue with them in more than one case. Unfortunately we are unable to give the details of these experiments, as the notes taken have been lost. In this connection we may mention that saline waters have been recommended in cases of mb. Menièri (Portmann).

Medicaments causing perspiration and salivation. Of these pilocarpin has been much resorted to for inner ear affections ever since Politzer[3] recommended it in 1880. We have not made many observations ourselves, as there exist numerous statistics and opinions as to the action of this medicament. (The majority of our 12 observations — Nos. 4, 13, 14, 17, 18, 51, 52, 56, 57, 66, 79, 82 — belong to former observations). In a few cases (Nos. 3, 14) we examined the hearing during the action of an injection and found the same result as Schubert, namely that hearing first grew worse and buzzing more severe, after which hearing increased. Here again we meet with the same two phases as with diuretics. In this connection we may mention, that Szász[1] has measured the pressure in the labyrinth during the action of pilocarpin, and also met with two phases, the first on rise of pressure, the second on fall of pressure.

Numerous otologists as Schubert. Moos[1], Oscar Wolff[2], L. Jacobson, Schirmunsky, Zawiska and J. Frotzl speak in favour of pilocarpin, whilst many also are against it. Zawiska recommends it in chronic and acute middle-ear troubles, which embrace the labyrinth and, owing to »tympanogen hydrops of the labyrinth», cause increased labyrinthal pressure. From our department Hvidt has published an article on treatment with pilocarpin, embracing 22 cases of Mb. Menièri among others (partially included in the present material). As some of Hvidt's pts were at the same time treated with catheterization, he finds it difficult to decide how much of the effects were due to pilocarpin and how much to catheterization. Hvidt, however, believes that he is justified in saying that a distinct effect was obtained in several of the cases. Pilocarpin was most active in fresh cases, and, where hearing increased, a distinct giddiness arose during treatment. A few statisticians as J. Frotzl give an improvement in hearing in 60—80 % of 53 cases.

In pilocarpin treatment output of fluid takes place by means of perspiration as well as by salivation. In our opinion there is no reason to suppose that there is any question of specific action on the ear any more when pilocarpin than when diuretics are used, but only of an increased output of fluid. It is thus easy to understand that the improvement in hearing is not maintained, and that the treatment has to be repeated when the depots of fluid are replaced.

Where pilocarpin produces no effect the reason in many cases is, no doubt, that perspiration has been slight, or that the output of fluid is immediately covered by fresh intake. This agrees with Schubert's advice, that the pt. should only drink very little after a pilocarpin cure.

Lucae[1] (p. 327) mentions a case in which hearing rose although there was no perspiration, but, he adds, there was a very great salivation. In one case (No. 3) we tried whether we by means of pilocarpin could produce perspiration although pt. had difficulty in perspiring, even in incandescent light bath. We did not attain our end and there was but sligt salivation, neither did hearing improve.

The effect of pilocarpin on hearing seems then, like that of the above mentioned diuretics, to be proportionate to the output of fluid.

Other medicaments, such as thiosinamin = allylthiourea and fibrolysin have been warmly recommended for inner ear complaints and soon again given up. Their action is possibly similar to that of the above mentioned. Löwensohn and Hirschland mention such experiments and Sugàr, in a synopsis, says that diuresis increases. There is then an increase of fluid output. The reason, perhaps why thiosinamin and fibrolysin do not come up to expectations is, that they are not sufficiently powerful fluid expelling agents, or that the pt. has immediately made up for the fluid lost. We ourselves have not experimented with these medicaments.

Baths are another means by which output of fluid can be effected. There exist reports as to the improvement of ear affections after mud-baths. E. Maljutin states that these baths influence tubal stenoses favourably.

We have tried *incandescent light baths* in a number of cases (for inst. Nos. 3, 14, 51, 59), but the effects of this treatment cannot be judged of with sufficient certainty, as other treatments, such as massage, gymnastics, reduced salt and fluid intake, were employed at the same time.

It may be surmised that the perspiration, running, dancing, walking and heat which pts themselves have observed as causing improvements of hearing, produce this result by means of an increased fluid out put.

On the contrary, in such cases where the pts have observed a decrease of hearing either from an increased fluid intake (soup, beer, sodawater) or from circumstances causing a decrease of evaporation (cold, damp air), we may surmise that the reason is the deposition of fluid.

Decreased fluid out put. This fits in with the circumstance, that in salyrganexperiment No. 77 we find increase of weight and decrease of hearing. In exp. 73 and 74 the pt. had a fluid intake greater than the output and the hearing remained unaltered or became, perhaps, slightly worse. This is most distinctly seen in experiments 68 and 72, where the intakes are large and, in spite of increasing diuresis, weight remains unaltered and hearing is worse or unaltered.

In several experiments (3, 5, 6, 21) we find the hearing, which

has improved after the injection, going down again as soon as weight increases.

Increased fluid intake. In 2 cases we made the experiment of letting pts (Nos. 3, 14) drink great quantities of fluid.

No. 3.

30/8 9.45 o'clock: v $\frac{20{,}00}{20{,}00}$ a_1M $\frac{16}{16}$ a_1A $\frac{52}{58}$ Li $\frac{18}{17}$ ÷ buzzings.

Besides the ordinary food 500 grs of milk were given at 10 o'clock and again at 14 o'clock.

17 o'clock: v $\frac{10{,}00}{10{,}00}$ a_1M $\frac{17}{17}$ a_1A $\frac{49}{47}$ Li $\frac{18}{20}$ Slight buzzings since 13 o'clock.

Diuresis $\frac{450}{1025}$ Fluid intake 1550 (from 30/8 8 o'clock —31/8 8 o'clock).

31/8 9 o'clock: v $\frac{4{,}00}{4{,}00}$ a_1M $\frac{16}{15}$ a_1A $\frac{45}{55}$ Li $\frac{24}{24}$ Increasing buzzings.

17 o'clock: v $\frac{4{,}00}{4{,}00}$ a_1M $\frac{18}{18}$ a_1A $\frac{44}{48}$ Li $\frac{24}{22}$ Buzzings, greatest on r.

Attacks of giddiness, nausea and violent buzzing in course of evening.

Diuresis $\frac{900}{1015}$ Fluid intake 2485.

1/9 9 o'clock: v $\frac{1{,}00}{1{,}25}$ a_1M $\frac{15}{16}$ a_1A $\frac{51}{50}$ Li $\frac{28}{28}$ (a_1M hardly as sonorous as otherwise).

Diuresis $\frac{1250}{1017}$ Fluid intake 2175.

2/9 8 o'clock:

2/9 8 ½ o'clock: v $\frac{1{,}00}{0{,}75}$ a_1M $\frac{14}{16}$ a_1A $\frac{48}{44}$ Li $\frac{24}{24}$ + buzzings.

No. 14.

29/8 20 o'clock: v $\frac{0{,}2—7{,}00}{15{,}00—20{,}00}$ a_1M $\frac{\text{vbr.}}{\text{vbr.}}$ a_1A $\frac{20}{20}$ Li $\frac{36}{28}$ ÷ buzzings. 500 grs of sodawater extra.

22 o'clock: v $\frac{0{,}1—6{,}00}{20{,}00}$ a_1A $\frac{12}{20}$ Li $\frac{30}{38}$ Violent buzzing for last ½ hr.

Diuresis 700 intake 1125 (from 29/8 8 o'clock —30/8 8 o'clock).

30/8 9 o'clock: v $\frac{aa}{aa}$ a_1M $\frac{0}{0}$ a_1A $\frac{10}{6}$ Li $\frac{90}{90}$

Both experiments show that the fluid intake was greater than the diuresis, also that hearing decreased only a few hours after the fluid was drunk.

Besides this we may refer to pt. no. 54. Here we ordered ½ ltr. of water extra a day, but were obliged to give it up, as she got worse buzzing, worse hearing also nausea and giddiness (with nystagmus).

The following observations may be added to the above. No. 100 declares that headache and buzzing return if she increases her intake of fluid. Pt. no. 58 had, on account of too low weight and tiredness following on an operation for hernia, put himself on a fattening diet which, besides the usual meals, consisted of the following:

6	o'clock	250	grs of chocolate.
7	»	150—200	grs of coffee.
9	»	1000	grs of jersey milk (+ lunch).
10	»	250	grs of bouillon.
12	»	350	grs pilsener ale (+ dinner).
15	»	250	grs of bouillon.
18	»	350	grs double ale (+ supper).
21	»	150—200	grs of coffee or tea.
total	about	2800	grs. (+ ordin. meals)

After having kept on this diet for 2 months, he suddenly one morning had a violent attack of giddiness although he had never been giddy before. After »draining» treatment the vestibular phenomena disappeared, and the greatly reduced hearing became normal in sympathy with a simultaneous loss of weight.

No. 134 increased his weight after discharge from hospital by 3 kilos in the course of 8 days, and by 7 kilos in 2 months. Simultaneously hearing decreased, buzzings reappeared and he again became giddy. No. 121 increased 4 kilos in the first 3 days after leaving hospital and became again giddy. No. 99 increased 2 kilos in 3 weeks, and buzzing, deafness and giddiness reappeared.

The majority of these increases in weight, which are only given as examples, are so great and rapid, that they can only be explained by a deposition of fluid, not of fat.

Influence of hydrostatic pressure. Many of the pts exhibit — as before mentioned — diurnal changes, hearing worst in the morning, when also their buzzings and headache are worst; some

have at the same time closing of the nose. It would seem obvious to consider this as dependent upon the hydrostatic pressure changing in unison with the change of posture. This supposition is strengthened by the fact, that some of these pts (Nos. 14, 37, 54, 105) get swollen legs in the evening, at the time when they hear best.

Our experiments and clinical observations have, then, proved that hearing can be influenced by manipulation of the in- and output of fluid of the whole body. The decrease of hearing in Menière-pts may therefore be supposed to depend upon there also being an abnormal fluid content in the ear, i. e. depot of fluid.

In literature we find like observations. Grahe[1] states, that hearing improves in nephritis pts simultaneously with the disappearance of the oedema, (8 cases exhibit loss of weight and better hearing, 1 loss of weight and unaltered hearing). S. Moos[2] reports intermitting deafness and buzzings which increased and disappeared in proportion to the diuresis and to the degree of anasarca and oedema of the face.

Rosenstein (moos[2] l. c.) likewise states, that hearing in nephritis pts may change from complete deafness to »voller Feinheit» and again to complete deafness, in accordance with decreasing and increasing oedema.

Lucae[1] (l. c. p. 326) relates of Banting — who was deaf as well as extremely stout — that his hearing improved simultaneously with a loss of weight of 35 lbs in 38 weeks; Lucae[1] (l. c. p. 82) also mentions decrease in hearing after meals. Itard (l. c. p. 334) does the same, and he moreover has noticed that hearing may grow worse in the course of the night. Schmalz who examined 951 deaf pts, found that 117 heard worst in the morning, 66 heard worst at night (l. c. p. 102) and 768 heard worst in damp weather (l. c. p. 101). V. Urbantschistch[2] speaks of a pt. with changing hearing, who was deaf at certain times of the day. He therefore supposes the case to be one of hysteria; Gradenigo[3] is of a corresponding opinion in like cases, whilst Gruber[3] and Jones Macnoughton warn strongly against such an interpretation.

Lucae[1] (l. c. p. 82) also mentions pts who heard better after dancing. Troeltsch[1] relates a case, where the pt's hearing

grew worse and buzzings increased if he only put on a cold boot. Gruber[1] reports on a pt., who heard badly in damp weather. Sharp tells us of a pt. who grew very deaf from drinking great quantities of tea from 8 to 10 times a-day; as soon as she gave this up her hearing increased and became almost normal, but grew worse again when, in spite of warning, she again began the bad habit.

Also these observations may find their natural explanation if we in analogy with our experiments, suppose them to be the result of changes in the fluid metabolism of the whole organism.

Interpretation of the results of the acoustic tests, especially with regard to boneconduction.

The question now arises — in what part of the auditory organ are we to seek the abnormal condition?

Do the abnormalities found in the acoustic function point to an affection of the sound transmitting or of the sound perceiving apparatus?

In the sound transmitting apparatus we include external ear, middle ear, fenestræ and labyrinthal fluid, in the sound perceiving the organ of Corti, n. cochlearis and the central nuclei and pathways, making use of the classification proposed by Rejtö.

Whisper. A reduced hearing of whisper (or conversation) may as well be due to a disease in the sound transmitting as in the sound perceiving apparatus, and gives in itself, therefore, no information as to the locality of the affection.

Airconduction. The same holds good with regard to airconduction.

Upper limit. In the greater number of cases of Mb. Menièri upper limit is found to be not at all, or only slightly, lowered. This seems to make against the supposition that we here have to do with an affection of the nerve, all the more so because Mb. Menièri may last for many years without giving a lasting defect in hearing.

In old and severe cases, where upper limit is lowered and hearing on the whole at the same time extremely bad, the nerve, at this advanced period is necessarily affected, but this must be considered as a secondary condition.

Lower limit. A raising of lower limit is considered by most as being a sign of an affection of sound transmission. It is often observed in diseases of the external ear, in tubal stenosis, suppuration of the middle ear, otosclerosis etc.

It has, however, been repeatedly maintained, here in Denmark by Thornval[1] with a reference to Siebenmann[1, 2, 3], Panse, W. Lange, Rosenstein, Schwabach, Engelhardt, Rehse and Holmgren that a raised lower limit is also characteristic of an affection of the nerve itself.

It is possible that an affection of the nerve may cause a reduction of the hearing as a whole, and thus reduce lower as well as upper limit, but that it should preferably involve the lower notes seems, at first sight, difficult to comprehend.

Neither can the greater part of the works quoted be considered as convincing. If we first of all consider those in which there are post-mortem examinations, we find, that Siebenmann's first report concerns a case where, besides acusticus-neuritis, stapes anchylosis was observed, whilst in the case in his other report special mention is made of the presence of changes in the middle ear, thickening of stapes and »embryonal», partially osseous tissue in the labyrinth. In Siebenmann's 3rd work we find 11 cases of tumour of the brain with deafness, of which only one is sufficiently examined acoustically.

In the cases described by Panse the tympanic membrane was »etwa normal» in the first, the bass deafness was not isolated, boneconduction was also annihilated, upper limit lowered and hearing of whisper but imperfect. As besides this the pt. understood conversation badly, and as no post mortem examination of the temporal bones was made, this case can neither be considered convincing. In two other cases the acoustic functional examination is wanting.

W. Lange has described 4 cases, in only the one of which a raising of lower limit was demonstrable, and in this case there were alterations of the tympanic membrane.

Rosenstein's pts are deficiently examined, there is no demonstrable bass deafness, and there are no post mortem examinations. Rhese mentions a case of tumor in the cerebellopontine angle without post mortem examination, but with

operation. This case exhibited affection of both lower and upper limit.

Rhese states distinctly that nuclear affection should be taken into consideration when a case of nervous deafness exhibits heightened lower limit, if *changes in the middle ear can be excluded.*

Grahe[2] has quite recently declared, on the basis of 3 clinically and pathologically-anatomically examined cases of acusticus tumors, that an affection of the nerve-stem is characterised by an elevation of lower limit.

Both elsewhere and here at the Municipal Hosp. raised lower limit, changed boneconduction and shortened airconduction have been seen with tumors, not only in the posterior but also in the anterior and middle fossa of the cranium. As suggested by S. H. Mygind[2], it would seem more reasonable to consider this bass deafness as being the result of a remote action of the tumour upon the labyrinth, especially upon the fluid system and upon the fenestræ, whose free mobility becomes impeded by the increased pressure on their medial surface, an effect which also can make itself felt on the »sound» ear. (Quix). We thus get a natural explanation why these pts so often present just the type of a middle ear disease. As soon as the mobility of the fenestra is impeded it will find expression in decreased hearing of whisper, shortened airconduction and elevated lower limit.

It may be supposed that, as in cerebral tumors, there is in mb. Menièri an increased pressure on the inner side of the fenestræ — a result, as appears from the previous chapter, of an increase of the fluid content in the labyrinth. As a change in the fixation can easily be produced, which is shown by the changing hearing not least of the lower notes, it would seem, that this fixation, which affects the transmission of sound, cannot be due to adhesions.

The same labile fixation can also be explained by a diminished pressure in the middle ear resulting from a tubal stenosis. An increase of the fluid content of the mucous membranes may account for such stenosis, as well as for a passing oedema of the middle ear.

In both cases we find a sufficient explanation of the raised lower limit, and there is therefore no reason for departing from the generally received opinion that raising of lower limit is due to an affection of the sound transmitting apparatus.

Boneconduction, which, in our material has exhibited all possible types, apparently renders the interpretation of the picture presented difficult. The circumstances that boneconduction in so many cases fluctuates in proportion to hearing of whisper and to lower limit, and in some cases is disproportionately short in spite of well preserved hearing, and in several cases exhibits great fluctuations before and after a momentary attack of giddiness, tempt, however, to a closer investigation of what an abridged boneconduction actually means.

Here we entered upon a field of dispute.

Cappivaccius, as early as 1603, drew attention to boneconduction. He investigated it by making a deaf person take the one end of an iron rod between his teeth, whilst the other end of the rod rested on a zither, which was struck. Cappivaccius thought, that if the pt. then could hear the note, this was a symptom of disease in the tympanic membrane, while if the pt. could not hear the note, the disease was to be sought for in the nerve. Already here the abridged or wanting boneconduction is used as a diagnostic of nervetrouble; as to the correctness of this postulate opinions have ever since differed.

Politzer[1], Bonnafont, Schmalz[1], Rinne, Erhardt, G. Alexander and many others think that an abridged boneconduction is a characteristic of nerve deafness.

It is also evident, that a diminuition of the perception of a note must involve the time of perception by boneconduction as well as by airconduction.

However, objections have been raised already many years ago against the supposition that an abridged boneconduction could be taken as evidence of an affection of sound perception, among others by Schwartze[1] and v. Troeltzsch. However, when v. Troeltzsch dispondently abandons the use of the tuning forks, the reason is primarily, that they do not ensure a knowledge even of what is heard and what is felt.

Schwartze[1] on the contrary, as also Lucae[5] and Gruber[3], quite clearly understood that an abridged, indeed extinguished boneconduction may be found both in middle and in inner ear affections.

Latterly several authors assert that abridged boneconduction

does not appear exclusively in affections of sound perception, but may also be met with in affections of sound conduction, namely when the round window cannot move to and fro normally. (Krainz[1], Rejtö[3], Biehl).

To us it appears also evident that every shifting in the relation between air- and boneconduction, whether resulting from a lengthened or a disproportionately abridged boneconduction, can only be the expression of an affection of conduction, as the note, when it once has reached the organ of perception, must be perceived in the same manner, regardless of the road by which it has travelled.

We will, for the present, disregard the fact that in a great number of cases it is difficult, or even impossible, to decide whether boneconduction is shortened or lengthened in proportion to airconduction, as the »decrement» of the note is different in branches and stem, and further as the period of boneconduction is in addition influenced by the pressure of the stem against the mastoid process.

We have, however, in a great numbers of cases found, on the one hand so considerable a lengthening of boneconduction coincident with relatively poor airconduction, and on the other hand (as above mentioned) a so considerable abridgement, indeed annihilation of boneconduction together with relatively good, indeed normal, hearing of whisper and a fairly good airconduction, that these cases, in our opinion, must be considered as exhibiting, in the one group a so evident lengthening and in the other a so evident genuine abridgement, that not only the lengthening but also the abridgement must be an expression of a pathologically changed sound transmission.

Thus, while boneconduction in its extreme genuine lengthening, as in its extreme genuine abridgement, can only be explained as being the expression of an affection of sound conduction, we can, as to the intermediate values, only say that they *may* be the expression of an affected sound transmission. That such a condition really exists as a rule, is proved by the elevated lower limit and well preserved upper limit, as mentioned above.

It is superfluous to substantiate that a lengthened boneconduction is the expression of an affection of sound conduction. The greatest lengthening is found, as well known, in stapes anchylosis,

both the secondary, resulting from inflammation of the middle ear and the primary, the genuine otosclerosis.

There are, however besides, other clinical observations which speak in favour of the opposite supposition, that an affection of sound transmission may cause abridgement of boneconduction. For inst. Goldmann and Lucae[2] have now and then observed abridgement of boneconduction in tubal stenosis, although a lengthening was here to be expected. In our material we also find several cases with abridged boneconduction, in spite of existing tubal stenosis, (Nos. 4, 6, 14, 15, 16, 37, 42, 74).

What do the experiments then prove?

Tourtual, Wheatstone and Weber, as we know, made observations as to boneconduction and found, that a watch placed between the teeth, or a tuning fork placed on the cranium, could be heard in both ears, and was heard better in the one ear when closed, for inst. with a finger.

Besides this Wheatstone examined boneconduction by filling the external meatus with water, the result being that boneconduction was lengthened. Quite recently Runge has repeated these water loading experiments and also found lengthening of boneconduction. Quicksilver-loading has given him exactly the same lengthening of boneconduction.

These experiments all prove that loading of the tympanic membrane with air, water or quicksilver gives lengthened boneconduction.

It is, however, interesting to see that the same influences may give an abridgement of boneconduction. This may be seen from Gellés experiment, by which he tries whether compression of air in the external meatus causes an abridgement of boneconduction or whether conduction remains unchanged. From the result obtained, he draws conclusion as to mobility or immobility of stapes. He thus connects abridgement with the mobility of stapes, that is with the sound conducting and not with the sound perceiving apparatus.

E. Bloch[2] produced abridgement or disappearance of boneconduction according to the force with which compression of air was applied in the outer meatus. Boneconduction was abridged already at a quicksilver pressure of 5—10 mm., and disappeared

at a pressure of 20—25 mm. He himself gives the explanation that an abridgement is due to pressure on the apparatus of the middle ear.

Mach[2], Lucae[2, 3], and Bezold[2, 3] examined how boneconduction was affected when the outer meatus was compressed by a finger. Mach[2] found that boneconduction was at first lengthened, but became abridged when pressure increased, disappearing at last entirely. Lucae[2, 3], found that pressure in the outer meatus increases boneconduction, but that an increasing pressure makes it again decrease somewhat, without, however, coming down to the normal value. Bezold[2] declares that boneconduction — tried on himself with fork a_1 — is diminished by pressure in the outer meatus; by degrees, as the pressure increases, boneconduction entirely disappears. Bezold[2] has also observed abridgement of boneconduction by aspiration in the external meatus and this surprised him, as he had anticipated lengthening, on account of the fixation of the ossicular chain.

From these experiments it is evident that manipulation of the sound conducting apparatus may cause abridgement of boneconduction. If we take the former experiments as evidence of a lengthened boneconduction being due to an affection of the sound conducting apparatus, we must also take the latter as evidence that an abridged boneconduction is due to the same. In these experiments it is impossible beforehand to prevent increased pressure occuring in the labyrinth. Now, one might suppose that such a heightened labyrinthal pressure would affect the sensorial epithelium and thus cause abridgement of boneconduction by means of an affection of sound perception, an idea which Politzer[1, 3] and Lucae[4] among others, have touched upon.

Bezold's experiment is against this, as he obtained abridged bone conduction, not only by compression, but also by aspiration — that is by means of the opposite conditions.

Krainz[1] by plugging fenestra rotunda — in pts with dry perforations and partially fixated ossicular chain — has also obtained abridgement of boneconduction together with synchronous improved hearing of whisper and lowering of lower limit. The latter circumstance speaks absolutely against the idea that such an eventual endolabyrinthal rise of pressure acts depreciatorily on

the perceptive apparatus, thus causing abridgement of boneconduction.

We have ourselves investigated the relation between air-and boneconduction with compression of air in the outer meatus. Whilst we thus in several cases of normal hearing found lengthened boneconduction we, in a single case, found that boneconduction was reduced and, when the pressure was raised, totally disappeared, whilst airconduction (determined by placing the stem of the tuning fork on a glass tube inserted in the middle of a 1 m long rubber tube, the one end of which is connected with the Politzer bag, the other with the ear) was comparatively unaffected.

Before experiment	a_1M (14) $\frac{-}{13}$	a_1A (25) $\frac{-}{25}$
Compression	a_1M (14) $\frac{-}{6}$	a_1A (25) $\frac{-}{21}$
Increased compression	a_1M (14) $\frac{-}{0}$	a_1A (25) $\frac{-}{18}$
Aspiration	a_1M (14) $\frac{-}{8}$	a_1A (25) $\frac{-}{21}$

This absolutely testifies against the supposition that the cause of the abridged or annihilated boneconduction is to be sought in an increased labyrinthal pressure, for in that case, airconduction would be correspondingly reduced.

But how can we explain the abridgement of boneconduction also being due to changes in the sound perceiving apparatus?

A review of the almost interminable problem of boneconduction lies outside the scope of the present work, neither can we enter into the details of the various theories, as to which we refer to Quix[1], Jørgen Møller, Frey[3] and others.

Many still consider Mach's theory as being the most satisfactory. This, the socalled »Abfluss Theorie» rests in principal on the idea that increased boneconduction depends upon a prevented escape of sound waves from the ear.

Bezold's »cranio-tympanal» theory has also many adherents. According to it, the boneconduction note is conducted to the labyrinth along 2 pathways, partly via tympanic membrane — ossicles — footplate, and partly via labyrinthal wall — ligamentum annulare — footplate. An increased tension or a fixation either of the ossicular

chain or the footplate in the window-frame will diminish the resistance to the transmission of the boneconduction note, and thus cause lengthened boneconduction.

Whilst these theories offer an explanation of the lengthened boneconduction, they are unable to explain an abridged boneconduction, as the authors themselves have been aware.

Mach[3], who in a letter to Politzer calls his own theory in question, is unable to bring it into harmony with the fact that he obtained abridgement, indeed even disappearance of boneconduction, by means of increased compression in the external meatus, which he considers due to tension of the tympanic membrane.

Bezold sees plainly that his theory does not explain the abridged boneconduction arising from his aspiration experiments.

There are, however, authors (Rejtö[3], Krainz[2]) who endeavour to explain how an affection of sound conduction can give rise to abridged boneconduction. Rejtö[3] thinks that the boneconduction sound is transmitted directly to the contents of the labyrinth through the labyrinthal wall. A defective mobility of stapes will cause the waves to be directed with greater energy from the osseous wall against fenestra rotunda, and thus give rise to increase of boneconduction. If, however, there is a diminished mobility of fenestra rotunda, the oscillations in the contents of the labyrinth will be weakened and boneconduction thus become shorter. Krainz[2] also thinks that defective mobility of fenestra rotunda may cause abridgement of boneconduction. His theory, to which Herzog[2] subscribes, is based upon phase displacement between vibrations transferred direct through the bone and vibrations transferred via the ossicular chain and footplate.

To both Rejtö[3] and Krainz[2] the objection may be made, that, as it is universally agreed that every diminuition of mobility, no matter in which fenestra, must cause abridged airconduction, neither of these authors' theories give any explanation of how there at the same time can be well preserved airconduction and disproportionally abridged, eventually annihilated, boneconduction.

Besides, Brünings has proved by clinical experiments, that a phase displacement between oscillations of the same note partly conducted via labyrinthal wall and partly conducted via

ossicular chain and footplate, does not produce any reduction of perception. This discovery, which »surprised» him greatly, has therefore led to his abandoning his former hypothesis of boneconduction, which was based on just such a labyrinthal interference.

We cannot see otherwise than that none of these hypotheses are able to explain a boneconduction which is abridged in relation to airconduction.

We may add, that some few authors, (Z i m m e r m a n n and B e c k m a n n), think that the ossicular chain does not serve in the transmission of the note but only plays a part in the regulation of the labyrinthal pressure, a hypothesis which, however, has not won much sympathy.

In our opinion such interpretations are not necessary, as B e z o l d's cranio-tympanal theory, which has always had the majority of adherents from the side of the clinic, is well able to explain all the boneconduction phenomena, also the disproportionate abridgement, if we modify it in one single point or, rather, carry it forward a few steps.

As mentioned above, B e z o l d's theory points to 2 pathways by which the boneconduction note may be transmitted to the organ of perception, the one is via the tympanic membrane, ossicular chain and footplate, the other via the lateral labyrinthal wall, ligamentum annulare and the footplate.

If any change takes place in one of these roads, boneconduction must also change. This is beautifully demonstrated in the well known comparison drawn between the human ear, which is principally designed for hearing by airconduction, and a whale's ear, which is contrived for hearing in water, that is to say by boneconduction. The ossicles of the whale are therefore clumsy, anchylotic and the surrounding cranial osseous tissue is compact; the stapes is also clumsy and massive and the edge of the footplate is brought into close contact with the window frame by means of cartilaginous tissue. (G. B o e n n i n g h a u s).* Every thing has been done to bring about first rate boneconduction and to prevent

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* *Postscript.* We have had the opportunity of examining a fresh specimen of a porpoise. The stapes was absolutely fixed in the oval window, but contrary to the above description the ossicles were not at all clumsy and seemed otherwise just as moveable as in human beings.

isolation. In the human ear boneconduction is, however, relatively but slightly developed, man being formed for hearing by airconduction. In order to prevent boneconduction acting disturbingly on air conduction and thus preventing the perception of sound, a sufficiently poor conduction from the skull, both through the cranium and through the ossicles, has been provided. This is easily obtained as far as the ossicular chain is concerned by the laxity of the connection between the single ossicles, and between these and the skull. In the case of the lateral labyrinthal wall the elastic ligamentum annulare forms an isolating, contact reducing connection between the footplate and the window frame.

If the fixation of the ossicular chain is increased (for inst. by a retraction of the tympanic membrane), or if the isolation around the footplate is destroyed, either because ligamentum annulare is replaced by otosclerotic tissue or by the formation of other connecting and conducting formations, boneconduction becomes lengthened. But, vice versa, we must from this deduce that when the isolation between stapes and the labyrinthal wall is increased, boneconduction is abridged.

How can we bring about an increased isolation and diminished contact between stapes and the labyrinthal wall?

Bezold[1] has measured the mobility of the fenestra in preparates belonging to normal individuals, and proved that the footplate may be displaced both in a medial and lateral diection; most however in lateral, the proportion being 1: 2.85.

The individual experiments proved that the relation between medial and lateral movements in relation to the wall of the labyrinth were always such, that the greatest displacement was lateral, but the experiments also proved, that in some cases the displacements were but slight, in others comparatively considerable.

The consequence we deduce is, that the displacement of stapes may bring about a less ideal contact with the window frame; the greater the displacement the lesser the contact, which again means a poorer, an abridged boneconduction.

Can this hypothesis give us an explanation of the existing facts?

The well known condition, that otosclerosis causes lengthened boneconduction, is easily understood, for here we have the isolation destroyed, and replaced by a broad, conducting connection

between stapes and the wall of the labyrinth. That lengthened boneconduction is, as a rule, found by inflammation in the middle ear, is explained by a lesser laxity in the connection of the ossicular chain. Adhesions to the surroundings may also occur, eventually a soldering fast of the hammer head to the roof of the tympanic cavity (R u n g e). In more severe cases the footplate may be soldered fast in the window frame; here then we have also an increase of contact. All the values of boneconduction may be found in cases of tubal stenosis. It is the different position of stapes, eventually combined with fixation of the ossicular chain, that is of importance in the individual cases.

By compressing air in the external meatus, boneconduction varies according to the mobility of the stapes and to the amount of pressure employed in each individual case. In M a c h's above mentioned experiment, boneconduction was lengthened by slight pressure, which corresponds to a fixation of the ossicular chain without any dislocation of the footplate. Greater pressure results in shorter boneconduction, which answers to stapes now relinquishing its ideal contact with the window frame and standing in medial position to the latter. A still further pressure, by which stapes is still more medially displaced, will give rise to a poorer contact, which corresponds to the disappearance of boneconduction.

L u c a e's[2, 3], and B e z o l d's[2] experiments with compression in the external meatus which seemingly give such opposite results, are not in reality at variance with one another. In L u c a e's experiment compression has not brought about any greater displacement of stapes, perhaps, even produced a slightly more intimate contact and, besides, fixation of the ossicular chain, consequently increased boneconduction. With B e z o l d stapes has evidently been displaced in a medial direction on compression, and in a lateral on aspiration, contact between stapes and the wall of the labyrinth in both cases becoming feebler, with the consequence that boneconduction has disappeared. The fixation of the ossicular chain has not been sufficient to counteract this. The varying boneconduction results, corresponding to the pressure employed in experiments with loading, as shown by E. B l o c h among others are thus also comprehensible. The fixation of the ossicular chain will find expression when the loading is slight and show itself

by a lengthened boneconduction, on heavier loading stapes will become displaced medially, which will be shown by an abridgement of boneconduction.

The above hypothesis coincides also with Politzer and Mader's experiments, in which they, by interrupting the ossicular chain, for inst. by sawing through the hammer-anvil articulation or the anvil-stirrup articulation, produced abridged boneconduction. It is possible that in these experiments, as on the whole, the degree of fixation of the ossicular chain is less decisive than the simultaneous change in the position of stapes in the window frame (ɔ: change of contact).* It is, however, difficult to separate the influence of these two factors.

If we wish to avoid calculating the share played by the ossicular chain in boneconduction, and to make a closer examination of the condition of stapes, we must make our experiments on patients radically operated upon.

Ferreri placed Lucae's pressure probe direct on stapes and found that boneconduction became abridged in proportion to the increase of pressure. Siebenmann found a lengthening of boneconduction by slight pressure on stapes, both which results agree with our theory.

We ourselves have examined cases of radical operation, partly with quicksilver loading and partly with water loading.

Quicksilver loading in patients radically operated upon. In 16 cases of quicksilver loading in the external meatus, we found that boneconduction remained unaltered in 8 cases, in 6 cases was lengthened, and in 2 abridged. In the cases of abridgement we must suppose that the footplate has given way slightly medially to the labyrinthal wall, whilst in those which exhibited lengthening, we may believe that the loading has made the footplate take up an ideal position to the labyrinthal wall; in such cases the foot plate must originally have been in a slightly lateral position. In one case boneconduction was extended to the double length. (J. 602/26: 18″—35″) (see table III).

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* *Postscript.* The principal importance of the pathway via labyrinthal wall-footplate seems to agree with the condition developed in the porpoise above described by us (moveable ossicular chain, fixed footplate).

We examined 14 of the same pts. by means of water loading (see table III) and only obtained one case of lengthening of boneconduction and one of abridgement, the rest remaining unaltered. This further proves that the change of boneconduction is conditional to the amount of pressure used.

TABLE III.

Experiments with Quicksilver and Water Loading on Radically Operated Patients.

($a_1M = 15$; $a_1A = 60$)

1.

556/26. ÷ suppuration.

Before experiment	$a_1M \frac{-}{13}$	$a_1A \frac{-}{18}$	$Li \frac{-}{55}$	$v \frac{-}{0}$
With quicksilver loading	$a_1M \frac{-}{13}$	$a_1A \frac{-}{11}$	$Li \frac{-}{55}$	$v \frac{-}{0}$
Without loading	$a_1M \frac{-}{13}$	$a_1A \frac{-}{18}$	$Li \frac{-}{55}$	$v \frac{-}{0}$
With water loading	$a_1M \frac{-}{13}$	$a_1A \frac{-}{14}$	$Li \frac{-}{55}$	$v \frac{-}{0}$
Without loading	$a_1M \frac{-}{13}$	$a_1A \frac{-}{18}$	$Li \frac{-}{55}$	$v \frac{-}{0}$

2.

523/26. ÷ suppuration. Cavity perfect.

Before experiment	$a_1M \frac{-}{18}$	$a_1A \frac{-}{25}$	$Li \frac{-}{36}$	$Ls \frac{\quad}{14000}$	$v \frac{\quad}{0.50}$
With quicksilver loading	$a_1M \frac{-}{17}$	$a_1A \frac{-}{14}$	$Li \frac{-}{36}$	$Ls \frac{\quad}{14000}$	$v \frac{-}{0}$
Without loading	$a_1M \frac{-}{18}$	$a_1A \frac{-}{24}$	$Li \frac{-}{36}$	$Ls \frac{\quad}{14000}$	$v \frac{\quad}{0.50}$
With water loading	$a_1M \frac{-}{18}$	$a_1A \frac{-}{20}$	$Li \frac{-}{36}$	$Ls \frac{\quad}{14000}$	$v \frac{-}{0}$
Without loading	$a_1M \frac{-}{18}$	$a_1A \frac{-}{25}$	$Li \frac{-}{36}$	$Ls \frac{\quad}{14000}$	$v \frac{\quad}{0.50}$

3—4.

705/24. Doublesided. Aur. dx.: Cavity dry, epidermised. Aur. sin.: Some secretion.

Before experiment	$a_1M \frac{18}{25}$	$a_1A \frac{33}{20}$	$Li \frac{24}{90}$	$v \frac{0.50}{0.50}$

With quicksilver loading $a_1M \frac{18}{19} a_1A \frac{20}{0} Li \frac{24}{150}$ $v \frac{0}{0}$

Without loading $a_1M \frac{20}{19} a_1A \frac{34}{18} Li \frac{24}{100}$ $v \frac{0.50}{0.50}$

With water loading $a_1M \frac{19}{19} a_1A \frac{24}{10} Li \frac{24}{100}$ $v \frac{0}{0}$

Without loading $a_1M \frac{18}{20} a_1A \frac{30}{10} Li \frac{24}{100}$ $v \frac{0.50}{0.50}$

5.

726/24. Aur. s.: Cavity epidermised, dry.

Before experiment $a_1M \frac{11}{14} a_1A \frac{32}{12} Li \frac{24}{90}$ $Ls \frac{17000}{15500}$ $v \frac{20.00}{0.40}$

With quicksilver loading $a_1M \frac{24}{14} a_1A \frac{17}{0} Li \frac{24}{>150}$ $Ls \frac{16000}{15000}$

Without loading $a_1M \frac{11}{11} a_1A \frac{32}{12} Li \frac{24}{90}$ $Ls \frac{17000}{15500}$

With water loading $a_1M \frac{22}{12} a_1A \frac{20}{0} Li \frac{24}{110}$ $Ls \frac{17000}{15000}$

Without loading $a_1M \frac{12}{12} a_1A \frac{30}{12} Li \frac{24}{90}$ $Ls \frac{17000}{15500}$

6.

173/15. Cavity perfect.

Before experiment $a_1M \frac{-}{17} a_1A \frac{-}{22} Li \frac{-}{36}$ $Ls \frac{}{16000}$ $v \frac{}{3.00}$

With quicksilver loading $a_1M \frac{-}{15} a_1A \frac{-}{12} Li \frac{-}{90}$ $Ls \frac{}{15000}$ $v \frac{-}{0}$

Without loading $a_1M \frac{-}{17} a_1A \frac{-}{23} Li \frac{-}{36}$ $Ls \frac{}{16000}$ $v \frac{}{3.00}$

7.

193/27. Cavity epidermised, dry.

Before experiment $a_1M \frac{19}{-} a_1A \frac{8}{-} Li \frac{90}{-}$ $Ls \frac{12000}{-}$ $v \frac{aa}{-}$

With quicksilver loading $a_1M \frac{19}{-} a_1A \frac{0}{-} Li \frac{>150}{-}$ $Ls \frac{9000}{-}$ $v \frac{0}{-}$

Without loading $a_1M \frac{19}{-} a_1A \frac{8}{-} Li \frac{90}{-} Ls \frac{12000}{---} v \frac{0}{-}$

With water loading $a_1M \frac{19}{-} a_1A \frac{0}{-} Li \frac{110}{---} Ls \frac{10000}{---} v \frac{0}{-}$

Without loading $a_1M \frac{19}{-} a_1A \frac{8}{-} Li \frac{90}{-} Ls \frac{12000}{---} v \frac{aa}{---}$

8.

661/26. Aur. dx.: Normal tymp. membrane. Aur. sin.: Cavity epidermised. Slight secretion at the bottom.

Before experiment $a_1M \frac{10}{8} a_1A \frac{35}{14} Li \frac{24}{150} Ls \frac{14000}{14000} v \frac{20.00}{aa}$

With quicksilver loading $a_1M \frac{17}{7} a_1A \frac{0}{10} Li \frac{36}{>150} Ls \frac{14000}{14000}$

Without loading $a_1M \frac{12}{7} a_1A \frac{30}{12} Ls \frac{14000}{14000}$

With water loading $a_1M \frac{18}{6\frac{1}{2}} a_1A \frac{16}{11} Li \frac{36}{>150} Ls \frac{14000}{14000}$

9.

602/26. Cavity epidermised, dry.

Before experiment $a_1M \frac{-}{18} a_1A \frac{-}{15} Li \frac{---}{140} Ls \frac{---}{19000} v \frac{---}{0.30}$

With quicksilver loading $a_1M \frac{-}{35} a_1A \frac{-}{0} Li \frac{---}{140} Ls \frac{---}{19000} v \frac{-}{0}$

Without loading $a_1M \frac{-}{18} a_1A \frac{-}{15} Li \frac{---}{140} Ls \frac{---}{19000} v \frac{---}{0.30}$

With water loading $a_1M \frac{-}{19} a_1A \frac{-}{15} Li \frac{---}{140} Ls \frac{---}{19000} v \frac{-}{0}$

Without loading $a_1M \frac{-}{18} a_1A \frac{-}{15} Li \frac{---}{140} Ls \frac{---}{19000} v \frac{---}{0.30}$

10—11.

724/24. Doublesided. Aur. d.: Dry epiderm. Cavity. Aur. s.: Odourless membranes, which can only be partially removed.

Before experiment $a_1M \frac{20}{22} a_1A \frac{15}{29} Li \frac{150}{110} Ls \frac{15000}{13000} v \frac{0.10}{0.30}$

With quicksilver loading $a_1M \frac{28}{27} a_1A \frac{13}{5} Li \frac{>150}{>150} Ls \frac{15000}{13500} v \frac{0}{0}$

Without loading	a_1M	19/21	a_1A	15/27	Li	150/110			v	0.10/0.30
With water loading	a_1M	20/21	a_1A	15/10	Li	>150/>150	Ls	15000/13000	v	0/0
Without loading	a_1M	20/22	a_1A	15/30	Li	150/110	Ls	15000/13000	v	0.10/0.30

12.

640/24. Aur. d.: Some secretion.

Before experiment	a_1M	20/15	a_1A	35/15	Li	20/18	Ls	19000/20000	v	2.00/20.00
With quicksilver loading	a_1M	24/24	a_1A	15/15	Li	90/20	Ls	19000/20000		
Without loading	a_1M	18/15	a_1A	36/60	Li	20/20	Ls	19000/20000		
With water loading	a_1M	20/24	a_1A	20/26	Li	20/20	Ls	19000/20000		

13.

610/24. Aur. s.: Cavity dry.

Before experiment	a_1M	21/24	a_1A	42/14	Li	20/150	Ls	16000/16000	v	2.50/0.10
With quicksilver loading	a_1M	—/23	a_1A	—/0	Li	—/>150	Ls	—/16000	v	—
Without loading	a_1M	—/23	a_1A	—/0	Li	—/>150	Ls	—/16000		
With water loading	a_1M	—/23	a_1A	—/0	Li	—/>150	Ls	—/16000		

14.

1/26. Aur. s.: Slight suppuration. Giddy on instillation in ear.

Before experiment	a_1M	14/10	a_1A	—/10	Li	—/36			v	—/0.50
With quicksilver loading	a_1M	—/18	a_1A	—/6	Li	—/150			v	—/0
Without loading	a_1M	—/15	a_1A	—/10	Li	—/36			v	—/0.50
With water loading	a_1M	—/15	a_1A	—/10	Li	—/110			v	—/0
Without loading	a_1M	—/15	a_1A	—/10	Li	—/36			v	—/0.50

15.

531/26. Aur. s.: Cavity dry.

Before experiment	$a_1M \frac{-}{12}$	$a_1A \frac{-}{15}$	Li $\frac{-}{90}$	Ls $\frac{-}{17000}$	v $\frac{-}{0.40}$
With quicksilver loading	$a_1M \frac{-}{17}$	$a_1A \frac{-}{0}$	Li $\frac{-}{>150}$	Ls $\frac{-}{15000}$	v $\frac{-}{0}$
Without loading	$a_1M \frac{-}{11}$	$a_1A \frac{-}{16}$	Li $\frac{-}{90}$	Ls $\frac{-}{17000}$	v $\frac{-}{0.40}$
With water loading	$a_1M \frac{-}{12}$	$a_1A \frac{-}{0}$	Li $\frac{-}{>150}$	Ls $\frac{-}{15000}$	v $\frac{-}{0}$
Without loading	$a_1M \frac{-}{12}$	$a_1A \frac{-}{15}$	Li $\frac{-}{100}$	Ls $\frac{-}{17000}$	v $\frac{-}{0.40}$

16.

1371/27. Aur. d.: Dry epiderm. cavity.

Before experiment	$a_1M \frac{17}{-}$	$a_1A \frac{15}{-}$	Li $\frac{23}{-}$	Ls $\frac{18000}{-}$
With quicksilver loading	$a_1M \frac{10}{-}$	$a_1A \frac{10}{-}$	Li $\frac{70}{-}$	Ls $\frac{18000}{-}$
Without loading	$a_1M \frac{15}{-}$	$a_1A \frac{15}{-}$	Li $\frac{23}{-}$	Ls $\frac{18000}{-}$

R u n g e has previously made the same experiments. He found no alteration in boneconduction, what he very justly explains by the existence of a fixation of stapes, (a condition especially aimed at by the method of operation used on his patients).

We now better understand a series of circumstances which we have not previously mentioned. For inst. B i e h l reports on a case in which a pt., with a defect of the tympanic membrane, exhibited a little tumor in the niche of fenestra rotunda. If the tumor were pressed Weber became lateralised to the sound ear, but if the tumor were lifted Weber went to the diseased ear.

This exactly corresponds to K r a i n z'[1] loading experiments referred to above. On inward pressure on fenestra rotunda stapes, namely, gives way compensatingly in a lateral direction, thus loosening its intimate contact and causing abridged boneconduction, whilst the opposite movement in fenestra rotunda makes stapes move inward, reaching ideal contact, i. e. boneconduction is lengthened.

We now also comprehend why boneconduction values change on lumbal puncture, the labyrinthal pressure being diminished, so that the position of the footplate becomes altered in relation to the window frame.

Thus O. Beck[2] after lumbal puncture of syphilis pts. finds, that their boneconduction, which previous to puncture was short, becomes longer, and after a few hours or days again becomes shorter. This he explains as dependent upon a change of the labyrinthal pressure.

Georg Kerekes after performing lumbal puncture on 20 individuals observed abridged boneconduction after puncture in the 15. He has also performed venaesection on pts. with arterial hypertension and obtained improvement of boneconduction as well as of hearing of whisper.

To still further prove the correctness of our hypothesis we have endeavoured experimentally to imitate the pressumed displacement in a pneumatic overpressure chamber.

From examinations of caisson labourers, divers, balloonists and air men we know that atmospheric pressure affects the ear.

Investigators like Thost[1], Gustav Heermann[2], Boot, F. Alt[1, 2], Poli, Tomka, J. Swiatecki, Heller, Mager and von Schrötter have examined caisson labourers and shown what morbid conditions may arise both in the ear and the rest of the body. These labourers were, in the majority of cases, not examined whilst actually exposed to the abnormal pressure, but first after they were out of it. Heermann, however, has also examined pts. during exposure to a constantly increased pressure. Many of such pts., however, were often exposed to so great changes of pressure that hearing was permanently injured. None of the existing works, then, give us any account of examinations of hearing during the actual change of pressure.

We have made such investigations with our so-called bell experiments. The idea arose with S. H. Mygind, on whose initiative Chr. Hvidt, as early as 1924, commenced the undertaking. His investigations have not, however, been published and are not made use of in the following.

Bell experiments. The so-called bell is a pneumatic chamber contrived for overpressure. The individuals to be examined, are

examined otoscopically before being taken into the bell, and the absence of tubal stenosis is also ascertained.

The examiner places himself with the individual to be examined in the bell, to and from which air can be pumped to the degree of pressure desired, and in which the pressure can be maintained fairly constant at the degree desired.

The individuals examined were all familiar with tuning fork tests and had been repeatedly examined prior to the bell experiments. They were often allowed to sit in the bell without being examined, in order to accustom them to the varying pressure, to the noise from the engine and the unaccustomed surroundings. As the pressure alters continuously it is necessary to make do with one determination of both bone- and airconduction, instead of taking 3 each time as otherwise done.

Before pressure begins to rise the acoustic condition of the individual examined is determined: boneconduction, lower limit airconduction and upper limit, also hearing of whisper, only, however, when this latter is under 2 m's, the diameter of the bell. Pressure is then increased, the individual examined being instructed to swallow in order to ensure the air being driven into the middle ear. During the rise of pressure boneconduction, lower limit, airconduction, and upper limit are determined.

We have never employed more than a slight rise of pressure 100—150 mm and, on one occasion, 200 mm quicksilver, that having proved sufficient for our purpose. As soon as the desired rise is reached, pressure is maintained constant and fresh determinations are taken. After this, pressure is allowed to fall, and determinations of the acoustic conditions are taken at intervals of a few minutes. Sometimes pressure has been allowed to fall immediately on the cessation of the rise, without maintaining the increased pressure at a constant pitch.

During the rise of pressure, the pressure in the middle ear will exceed that in the labyrinth, during the fall the opposite takes place. In normal individuals, as the table shows, the following takes place: both during the rise and the fall of pressure abridgement of airconduction, elevation of lower limit and also a disproportionate abridgement of boneconduction are met with, the last especially during the fall, when it can reach 0, although airconduction is then rather better than during the rise.

TABLE IV.

Bell Experiments in Normal Individuals.

(When nothing particularly indicated $a_1M = 15''$, $a_1A = 60''$.)

No. 1.

Rise 150 mm in 20 min. Up for 15 min. Fall in 15 min.

4/6 26 M. M. 42 years	Before experiment	Beginning of rise	2 min. later	5 min. later	C. 10 min. later	C. 15 min. later	Up	Up 6 min.	Up 10 min.	Up 15 min.	Beginning of fall	2 min. later	4 min. later	10 min. later	15 min. later	Down	5 min. Down
a_1M (10)	9/9	—		2.2/2.2			c. 2/c 2		2/2	-	0/0		0/0		0/0	6/5	
a_1M (22)	16/16	3.4/3.4	3.4/3.4		4/5		3.4/3.4	6/5	7/7	6/5	3/3	?/0	0/0		0/0	16/14	
a_1A (25)	25/25	10.20/20			1/—			19/14		13/12				8/8			20/21
a_1A (105)	—/105	—			18/20				45/55	45/55		14/10	25/30		25/37	85/95	
Li	15/15	90/90		90/90		90/90			28/28	26/26			45/45	36/36			24/26
v	20.00/20.00													1.00/1.00			
Remarks				Pain in l. ear.		Auto-phonia		Pain ceasing						Auto-phonia	Press in ears		

No. 2.

Rise 150 mm in 15 min Up for 30 min. Fall in 15 min.

18/4 26 M. M. 42 years	Before experiment	Beginning of rise	2 min. later	5 min. later	10 min. later	15 min. later	Up 15 min.	Up 30 min.	Beginning of fall	5 min. later	10 min. later	Down	2 min. Down
a_1M (10)	9/9	$4\frac{1}{2}.4$	$2.2\frac{1}{3}$	Vbr.	Vbr.		5.5			??	0	7	9.8.7.
a_1A (25)	27/26	10.12	$8\frac{1}{2}.7$	10	7—12					$15\frac{1}{2}$			28
Li	17/17			30		65/65	36				34		23
Ls	18000				16000/16000		16000				17000		17000
Remarks		Auto-phonia			Pain in l. ear	Press on both ears		No pressure			Pain in ears		No sensations.

No. 3.

Rise 150 mm in 15 min. Up for 15 min. Fall in 15 min.

1/6 26 F. H. 50 years	Before experiment	Beginning of rise	2 min. later	4 min. later	10 min. later	12 min. later	c. 14 min. later	Up	Up 15 min.	Beginning of fall	5 min. later	c. 10 min. later	12 min. later	Down
a_1M (10)	9.9 / 10.10	8 / 9	7 / 6	6 / 6	6 / 6	— / 4	4 Vbr.	— / 7	9 / 9	— / 6	— / 4	— / 4	3 Vbr.	— / 10
a_1A (25)	25 / 23	10 / 11	— / 12	—	— / 17	—	— / 15		— / 22		— / 15			— / 30
Li	18 / 18		— / 24			— / 30			— / 18		— / 22	— / 18	— / 22	—
Ls	17000 / 16000				14000				16000					16000
v	20.00 / 20.00				—									

No. 4.

Rise 100 mm in 15 min. Up for 20 min. Fall in 15 min.

15/6 26 F. M. 41 years	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	Up	Up 5 min.	Up 10 min.	Up 15 min.	Up 20 min.	Beginning of fall	5 min. later	10 min. later	15 min. later	Down	5 min. Down
a_1M (10)	— / 12	— / 5	— / 3.3	— / 3.3	— / 3		— / 6	— / 6	— / 6	— / 6	— / 6	— / 6	— / 8	— / 3.4		— / 11
a_1A (25)	— / 30	— / 15		— / 15		— / 15	— / 14	— / 16	— / 17	— / 17	— / 15		— / 15	— / 20	— / 25	— / 30
Li	— / 16		— / 20		— / 24		— / 22	— / 22	— / 19			— / 23		— / 19	— / 16	— / 16
Ls	15500 / 15000			15000						15200						15000
Remarks	÷ buzz.		÷ cracking			Press. in forehead										

No. 5.

Rise 100 mm in 5 min. Up for 5 min. Fall in 5 min.

2/6 27 M. W. 20 years	Before experiment	Beginning of rise	c. 1 min. later	c. 2 min. later	3 min. later	4 min. later	Up	Up 5 min.	Beginning of fall	1 min. later	2 min. later	c. 4 min. later	4 min. later	5 min. later	Nearly down	Down
a_1M	15/15	10/6	7	8	7/6	6	8+8	9	7	3	3.3	4	0	0	7	17
a_1A	55/57	25									25			25		
Li	15/15	24			24		24	36			24					
Ls.	18000/18000										18000					
Remarks	÷ buzzing ÷ giddiness ÷ headache															

No. 6.

Rise 100 mm in 5 min. Up for 5 min. Fall in 5 min.

2/6 27 M. V. 22 years	Before experiment	Beginning of rise	1 min. later	2 min. later	4 min. later	Up	Up 5 min.	Beginning of fall	1 min. later	2 min. later	3 min. later	c. 4 min. later	4 min. later	Nearly down	Down
a_1M	17/17	10	8	10	11	14	14	10/8	7	7	6.6	5	7	11	15
a_1A	60/60		25							25					
Li	15/15				24	15	15						24		
Ls	19000/19000							19000/19000							
v	20.20/20.20	÷ buzzing ÷ giddiness ÷ headache													

No. 7.

Rise 100 mm in 5 min. Up for 5 min. Fall

31/5 27 F. C. 18 years	Before experiment	Beginning of rise	1 min. later	3 min. later	4 min. later	Up	Up 2 min.	Up 4 min.	Beginning of fall	2 min. later	3 min. later	4 min. later	Nearly down	Down	5 min. down
a_1M	14/14	7	6.6	5	7.7	8	8.8	8.8	6	4.4	5	4.4	11	16	15
a_1A	56/52				40						38				
Li	15/15			24						24					
Ls	18000/18000														
Remarks	v. 20.00/20.00 ÷ headache ÷ giddiness ÷ buzzing														

No. 8.

Rise 100 mm in 5 min. Up for 5 min. Fall.

23/6 27 M. D. 48 years	Before experiment	Beginning of rise	1 min. later	3 min. later	4 min, later	5 min. later	Up	Up 5 min.	Beginning of fall	1 min. later	4 min. later	Nearly down	Down
a_1M	13/13	11	6	11	8	6	9	11	5	5	2	5	9
a_1A	56/55		60			40	38	45			41		55
Li	15/15			20		24	24			20			15
Ls	17000/17000			17000		17000	17000			17000		17000	
Remarks	v. 20.00/20.00 ÷ headache ÷ giddiness ÷ buzzing												

No. 9.

Rise 100 mm in 5 min. Up 5 min. Fall.

24/5 26 F. I. 39 years	*Before experiment	Beginning to rise	2 min. later	5 min. later	Up	Up 2 min.	Up 5 min.	Beginning to fall	2 min. later	3 min. later	5 min. later	Down	5 min. down
a_1M	11.11	7.7	6.6	6.6	9	6.6	8	6	5	$4\frac{1}{2}$	$4\frac{1}{2}$	10.10	11.11
a_1A	50	20		25	40						25		
Li	20			24					24				
Ls	17000				17000								17000
Remarks	÷ giddn. ÷ buzzing ÷ headache *Disturbing noise in the room. a_1A, Li, a_1M normal, when tested the next day												

No. 10.

Rise 100 mm in 10 min. Fall immediately on cessation of rise.

18/6 27 M. M. 43 years	Before experiment	2 min. after beginning of rise	3 min. later	5 min. later	6 min. later	7 min. later	8 min. later	10 min. later	Beginning of fall	1 min. later	2 min. later	3 min. later	c. $3\frac{1}{2}$ min. later	4 min. later	5 min. later	$5\frac{1}{2}$ min. later	5 min. down
a_1M	14	6	5	0	2	4	4.4	5.5	5	4	3	2.2	3	1	0	0	14
a_1A	61	—	—	17			24				17	20		20	22	—	
Li	15		20		24		30	20				30		24	—	—	
Ls	—	—	14000									14000		15000			

No. 11.

Rise 100 mm in 10 min. Fall immediately on cessation of rise.

18/6 27 F. H. 42 years	Before experiment	Beginning of rise	2 min. later	4 min. later	6 min. later	8 min. later	Up	Beginning of fall	1 min. later	2 min. later	3 min. later	3½ min. later	4 min. later	5 min. later	Down	5 min. down
a_1M	15	9	4	9	8	8	6	6.6	7	2.2	0	0	0	2	5	15
a_1A	57		22			18				17			15		22	
Li	15	15	24				24			24					20	
Ls		15000			14000						15000		15000			

No. 12.

Rise 100 mm in 5 min. Fall immediately on cessation of rise.

7/6 27 F. O. 20 years	Before experiment	Beginning of rise	1 min. later	2 min. later	3 min. later	4 min. later	Up	Beginning of fall	1 min. later	3 min. later	Nearly down	Down
a_1M	12	8	10	9	8	11	9	5	5	6	9	10
a_1A	48			30						40		44
Li	15				26				24			15
Ls	19000						19000					19000

No. 13.

Rise 100 mm in 15 min. Up. for 5 min. Fall.

31/5 27 F. G. 46 years	Before experiment	Beginning of rise	2 min. later	4 min. later	5 min. later	Up	Beginning of fall	2 min. later	3 min. later	4 min. later	5 min. later	Down
a_1M	14.14	10.10	7	0	5	10	7.7	Vbr.	Vbr.	Vbr.	6.7	14
a_1A	58											
Li	15											
Ls	16000											
Remarks	÷ Press. on ears ÷ giddin. ÷ headache ÷ buzz. No otoscopy, never had ear trouble.											

No. 14.

Rise 100 mm in 5 min. Fall immediately on cessation of rise.

30/6 27 M. K. 20 years	Before experiment	Beginning of rise	2 min. later	Up	Beginning of fall	2 min. later	4 min. later	Down
a_1M	14	6	6	5	5	4		12
a_1A	60		35		30	33		52
Li	15		20		24		24	17
Ls	18000		18000		18000		18000	18000

TABLE V.

Bell Experiments in Patients suffering from Otosclerosis.

No. 1.

Rise 150 mm in 15 min. Up for 10 min. Fall.

9/12 26 F. H. 52 years	Before experiment	5 min. after begin. of rise	10 min. later	5 min. constant pressure	10 min. const. pressure	Beginning of fall	5 min. later	Fall nearly finished	5 min. down
a_1M	$\frac{\cdot\cdot}{22}$	$\frac{}{20}$	$\frac{}{20}$	$\frac{}{21}$	$\frac{}{25}$	$\frac{}{21}$	$\frac{}{22}$	$\frac{}{24}$	$\frac{}{25}$
a_1A	$\frac{}{10}$	—	$\frac{}{5}$	$\frac{}{5}$	—	$\frac{}{7}$	—	—	$\frac{}{10}$
Li	$\frac{}{150}$	$\frac{}{150}$	$\frac{}{150}$	$\frac{}{150}$	$\frac{}{150}$	—	—		
Ls	$\frac{}{12400}$	$\frac{}{12400}$	$\frac{}{12400}$	$\frac{}{12400}$	$\frac{}{12400}$	$\frac{}{12400}$			$\frac{}{12400}$
Remarks	÷ giddin.		buzzing increasing			÷ headache			

No. 2.

Rise 150 mm in 20 min. Fall immediately on cessation of rise.

3/5 27 F. L. 33 years	Before experiment	Beginning of rise	7 min. later	14 min. later	17 min. later	20 min. later	Beginning of fall	5 min. later	10 min. later	15 min. later	5 min. down
a_1M	$\frac{16}{10}$	$\frac{13}{\cdot\cdot}$	$\frac{12}{\cdot\cdot}$	$\frac{16}{\cdot\cdot}$	$\frac{14}{\cdot\cdot}$	$\frac{17}{\cdot\cdot}$	$\frac{15}{\cdot\cdot}$	$\frac{15}{\cdot\cdot}$	$\frac{16}{\cdot\cdot}$	$\frac{16}{\cdot\cdot}$	$\frac{18}{\cdot\cdot}$
a_1A	$\frac{20}{8}$	$\frac{18}{\cdot\cdot}$	$\frac{18}{\cdot\cdot}$	$\frac{20}{\cdot\cdot}$	$\frac{20}{\cdot\cdot}$	$\frac{19}{\cdot\cdot}$	$\frac{22}{\cdot\cdot}$	$\frac{18}{\cdot\cdot}$	—	$\frac{18}{\cdot\cdot}$	$\frac{20}{\cdot\cdot}$
Li	$\frac{90}{150}$	$\frac{90}{\cdot\cdot}$	$\frac{90}{\cdot\cdot}$	$\frac{90}{\cdot\cdot}$	—	—	—	$\frac{50}{\cdot\cdot}$	$\frac{60}{\cdot\cdot}$	$\frac{50}{\cdot\cdot}$	$\frac{120}{\cdot\cdot}$
Ls	$\frac{8000}{\cdot\cdot}$	—	—	—	—	$\frac{8000}{\cdot\cdot}$	—	—	—	$\frac{8000}{\cdot\cdot}$	$\frac{8000}{\cdot\cdot}$
Remarks	÷ giddn.			buzzing not worse than usualy hearing not improved			÷ headache				

No. 3.

Rise 100 mm in 5 min. Up for 5 min. Fall.

28/5 27 F. S. 42 years	Before experiment	Beginning of rise	1 min. later	2 min. later	5 min. later	Up	2 min. up	Beginning of fall	2 min. later	5 min. later	Down
a_1M	$\frac{}{15}$	$\frac{}{18}$	$\frac{}{15}$	$\frac{}{15}$	$\frac{}{18}$	$\frac{}{15}$	$\frac{}{18}$	$\frac{}{14}$	$\frac{}{18}$	$\frac{}{18}$	$\frac{}{18}$
a_1A	$\frac{}{15}$	—	$\frac{}{8}$	$\frac{}{8}$	$\frac{}{8.8}$	—	—	$\frac{}{10}$	$\frac{}{10}$	—	$\frac{}{10}$
Li	$\frac{}{55}$	—	—	$\frac{}{90}$	—	$\frac{}{90}$	$\frac{}{90}$	—	—	—	—
Ls	$\frac{}{8000}$	—	—	—	—	$\frac{}{8000}$	—	—	—	—	$\frac{}{8000}$
Observat.	÷ giddin.	÷ increasing buzz.						÷ headache			

No. 4.

Rise 100 mm in 5 min. Fall immediately on cessation of rise.

1/7 27 F. J. 37 years	Before experiment	1 min. after beginn. of rise	3 min. later	Beginning of fall	3—4 min. later	Down
a_1M	$\frac{15}{19}$	$\frac{15}{19}$	$\frac{15}{19}$	$\frac{15}{19}$	$\frac{15}{19}$	$\frac{15}{19}$
a_1A	$\frac{18}{12}$	$\frac{14}{12}$	$\frac{13}{11}$	$\frac{13}{15}$	$\frac{15}{15}$	$\frac{13}{15}$
Li	$\frac{36}{40}$	$\frac{36}{40}$	$\frac{65}{40}$	$\frac{36}{36—40}$	$\frac{36}{40}$	$\frac{36}{40}$
Ls	$\frac{12000}{10000}$				$\frac{12000}{10000}$	

No. 5.

Rise 100 mm in 5 min. Fall immediately on cessation of rise.

23/6 27 F. O. 42 years	Before experiment	Beginning of rise	1 min. later	4 min. later	Beginning of fall	2 min. later	4 min. later	Down
a_1M	$\frac{}{20}$	$\frac{}{20}$	$\frac{}{20}$	$\frac{}{22}$	$\frac{}{20}$	$\frac{}{19}$	$\frac{}{19}$	$\frac{}{20}$
a_1A	$\frac{}{13}$		$\frac{}{10}$			$\frac{}{14}$	$\frac{}{20}$	$\frac{}{14}$
Li	$\frac{}{70}$		$\frac{}{90}$			$\frac{}{90}$		$\frac{}{70}$
Ls	$\frac{}{12000}$				$\frac{}{12000}$	$\frac{}{13000}$	$\frac{}{12500}$	$\frac{}{12000}$

No. 6.

Rise 100 mm in 5 min. Fall immediately on cessation of rise.

M. P. 44 years	Before experiment	Beginning of rise	2 min. later	4 min. later	Beginning of fall	4 min. later	Down
a_1M	$\frac{10}{}$	$\frac{10}{}$	$\frac{9}{}$	$\frac{9}{}$	$\frac{10}{}$	$\frac{10}{}$	$\frac{10}{}$
a_1A	$\frac{0}{}$			$\frac{0}{}$		$\frac{0}{}$	$\frac{0}{}$
Li	$\frac{70}{}$		$\frac{90}{}$		$\frac{110}{}$	$\frac{90}{}$	$\frac{90}{}$
Ls	Cannot be determined, as pt. declares he hears monochord even when not rubbed.						

No. 7.

Rise 160 mm in 15 min. Up 10 min. Fall.

17/5 27 M. M. 44 years	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	Up	Up 5 min.	Up 10 min.	Beginning of fall	5 min. later	10 min. later	12 min. later	Down	5 min. down
a_1M	22/16	16/17	17/17	22/18	21/17	/18	17/17	21/18	21/17	21/17	21/16	22/16	22/16	22/17
a_1A	18/18	/18	12/18	/16	10/18	—	8/17	15/15	12/21	12/21	17/25	17/25	20/22	18/30
Li	60/60	60/60	—	90/90	—	—	90/90	—	80/70	—	90/70	—	90/65	80/65
Ls	15000/16000	14000/16000	—	—	—	—	14000/15000	—	14000/15000	—	14000/15000	—	—	14000/15000
Remarks	÷ headache ÷ increased buzzing													

No. 8.

Rise 150 mm in 15 min. Fall immediately on cessation of rise.

7/6 25 F. H.	Before experiment	Beginning of rise	1 min. later	2 min. later	5 min. later	Up	Beginning of fall	2 min. later	5 min. later	Down
a_1M	/19	/21	/21	/20	/20	/19	/19	/20	/21	/21
a_1A	/23		/20			/23	/23			
Li	/90		/90			/90	—	/90		/90
Ls	/12000				/12000					/12000
Remarks	Testing of a_1A difficult on account of buzzing									

TABLE VI.

Bell Experiments on Menière-Patients.

($a_1M = 15''$ $a_1A = 60$, when not otherwise indicated).

N.o 1.

Rise 250 mm in 15 min. Up for 15 min. Fall.

14/12 Pt. No. 57	Before experim.	Beginn. of rise	5 min. later	10 min. later	Up	Up 5 min.	Up 10 min.	Up 15 min.	Beginn. of fall	5 min. later	10 min. later	15 m in. later	5 min. down
a_1M	12	5	0	0	5	7	9	9	5	5	4	3	21
a_1A	26	35	20	30	35	37		32	26	36	30	30	34
Li	90	90		90		80		80		55	60	60	55
Remarks	÷ giddiness				÷ headache						÷ buzzing		

No. 2.

Rise 150 mm in 15 min. Up for 15 min. Fall.

14/12 26 Pt. No. 8	Before experim.	Beginn. of rise	5 min. later	10 min. later	Up	Up 5 min.	Up 10 min.	Up 12 min.	Up 15 min.	Beginn. of fall	2 min. later	10 min. later	15 min. later	5 min. down
a_1M	6	0	0	0	3	6	0	4	6	3	0	0	0	6
a_1A	32	25	21	25	35	25	38	—	32	27	—	27	28	40
Li	States he hears unsounded tuning fork													

No. 3.

Rise 150 mm in 15 min. Up for 15 min. Fall.

10/12 26 Pt. No. 36	Before experiment	Beginning of rise	5 min. later	10 min. later	Up	Up 5 min.	Up 10 min.	Up. 15 min.	Beginning of fall	5 min. later	10 min. later	15 min. later	5 min. down
a_1M	$\frac{6}{12\frac{1}{2}}$	$\frac{5}{4}$	$\frac{0}{-}$	$\frac{0}{-}$	$\frac{5}{-}$	$\frac{1}{-}$	$\frac{3}{-}$	$\frac{3}{-}$	$\frac{0}{-}$	$\frac{0}{-}$	$\frac{0}{-}$	$\frac{0}{-}$	$\frac{10}{-}$
a_1A	$\frac{15}{50}$	$\frac{14}{-}$	$\frac{13}{-}$	$\frac{14}{-}$	$\frac{14}{-}$	$\frac{20}{-}$	$\frac{18}{-}$	$\frac{16}{-}$	$\frac{14}{-}$	$\frac{14}{-}$	$\frac{14}{-}$		$\frac{17}{-}$
Li	$\frac{70}{-}$	$\frac{70}{-}$	$\frac{70}{-}$	$\frac{70}{-}$	$\frac{55}{-}$		$\frac{55}{-}$	$\frac{55}{-}$		$\frac{60}{-}$	$\frac{60}{-}$		$\frac{60}{-}$
v.	$\frac{0.50}{-}$		$\frac{0.50}{-}$					$\frac{0.50}{-}$		$\frac{0.50}{-}$			
Remarks	÷ giddiness			÷ headache					÷ buzzing				

No. 4.

Rise 100 mm in 10 min. Fall immediately on cessation of rise.

10/5 27 Pt. No. 29	Before experiment	Beginning of rise	2 min. later	5 min. later	10 min. later	Beginning of fall	2 min. later	7 min. later	10 min. later	Down	5 min. later	10 min. later
a_1M	$\frac{}{9}$	$\frac{}{7}$	$\frac{}{3}$	$\frac{}{4}$	$\frac{}{4}$	$\frac{}{3}$	$\frac{}{2}$	$\frac{}{2}$	$\frac{}{0}$	$\frac{}{4}$	$\frac{}{7}$	$\frac{}{9}$
a_1A	$\frac{}{34}$	$\frac{}{25}$	$\frac{}{25}$	$\frac{}{25}$	$\frac{}{29}$	$\frac{}{28}$	$\frac{}{30}$	$\frac{}{25}$	$\frac{}{28}$	$\frac{}{46}$	$\frac{}{46}$	$\frac{}{50}$
Li	$\frac{}{21}$		$\frac{}{24}$			$\frac{}{24}$			$\frac{}{24}$		$\frac{}{24}$	$\frac{}{21}$
Ls	$\frac{}{15000}$		$\frac{}{16000}$		$\frac{}{14000}$		$\frac{}{16000}$		$\frac{}{16000}$		$\frac{}{17000}$	
Remarks	÷ giddiness			÷ increas. buzzing								

No. 5.

Rise 100 mm in 5 min. Fall immediately on cessation rise.

30/6 27 Pt. No. 43	Before experiment	2 min. after beginning of rise	5 min. later nearly up	Beginning of fall	2 min. later	4 min. later	Down
a_1M	7/7*	5/6	4/6	6/0	3/4	—	10/10
a_1A	20/22		15/10	15/15		15/10	24/28
Li	30/30		55/90			24/70	24/24
Ls	16000/17000		15000/15000			16000/17000	16000/17000
Remarks	* a_1M on l. side probably transfer from r. buzzing on l. ear, not on r. ÷ headache ÷ giddiness						

No. 6.

Rise 100 mm in 5 min. Up for 5 min. Fall.

23/6 27 Pt. No. 19	Before experiment	Beginning of rise	1 min. later	3 min. later	4 min. later	5 min. later	Up	Up 5 min.	Beginning of fall	2 min. later	4 min. later	5 min. later	Down
a_1M	15/15	6	13	5	5	5	5	10	6	6	9	6	11
a_1A	41/42		20			21	22	25			20		30
Li	15/15		20			20	20			20			15
Ls	15000			18000		18000	17000			17000		17000	

No. 7.

Rise 100 mm in 5 min. Up for 5 min. Fall.

23/6 27 Pt. No. 74	Before experiment	Beginning of rise	1 min. later	2 min. later	4 min. later	Up	Up. 5 min.	Beginning of fall	1 min. later	2 min. later	4 min. later	5 min. later	Down
a_1M	12	6	6	9	0	5	11	5	6	4	0	5	5
a_1A	42		30		15	19	25				11		30
Li	22			24	24	30				24			23
Ls	16000			18000	19000	17000				17000	17000	18000	17000
Remarks	+ Nausea. Nystagmus not looked for												

No. 8.

Rise 150 mm in 15 min. Up for 15 min. Fall.

3/5 27 Pt. No. 29	Before experiment	Beginning of rise	2 min. later	7 min. later	10 min. later	Up	Up 10 min.	Up 12 min.	Up 15 min.	Beginning of fall	5 min. later	10 min. later	15 min. later	5 min. down
a_1M	5	0	vbr.	vbr.	vbr.	vbr.	3	4	6	vbr.	vbr.	vbr.	3	11
a_1A	30	25	24	15	30	30	25	40		25	35			42
Li	34	36	34					34		34	34			24
Ls			13000				14000	13000		14000	14000			
Remarks	a_1M stated as vibration (vbr.), not perceived as note ÷ giddiness ÷ increased buzzing													

No. 9.

Rise 150 mm in 10 min. Up for 15 min. Fall.

25/1 27 Pt. No. 4	Before experiment	Beginning of rise	5 min. later	10 min. later	Up	Up 10 min.	Up 15 min.	Beginning of fall	10 min. later	5 min. down
a_1M	0	3	3	3	0	0	0	0	0	0
a_1A	0	0	0	6	0	5	3		0	0
Li	>150	>150			>150				>150	>150
Ls	9000		8000			11000			11000	9000
v.	0.05	0.10				0.10	0.10			0.05

No. 10.

Rise 100 mm in 5 min. Fall immediately on cessation of rise.

30/6 26 Pt. No. 51	Before experiment	Beginning of rise	4 min. later	Beginning of fall	3 min. later
a_1M	5 / 0	5 / 4	5 / 4	8 / 0	6 / 0
a_1A	15 / 0		10	12 / 0	
Li	32 / 100		90		70
Ls	8000 / 14000		8000		8000
v.	0.10		0.30		0.30

No. 11.

Rise 100 mm in 15 min. Fall immediately on cessation of rise.

3/4 26 Pt. No. 14	Before experiment	5 min. after beginning of rise	15 min. later	Beginning of fall	5 min. later	Down	1 hour down
a_1M (10)	1/2	3/3		0/0	0/0	0/0	vbr./vbr.
Li	36/36	90/50	50/40		40/40		34/38
Ls	14000/14000	13000/13000			13000/13000		13500/13000
v.	0.35/0.65		1.00/1.00			0.10/0.20	0.10/0.30
Remarks	÷ pressure. Feels well during rise. Pressure on ears during fall, also giddiness						

No. 12.

Rise 200 mm in 20 min. Fall immediately on cessation of rise.

3/5 27 Pt. No. 14	Before experiment	Beginning of fall	5 min. later	10 min. later	15 min. later	20 min. later	Beginning of fall	Down
a_1M	0/0	0/0	—/vbr.	vbr./vbr.	vbr./2	vbr./4	0/0	0/0
a_1A	—/10					—/10		
Li	—/90			—/80		—/36		—/90
Ls	—/15000			—/15000		—/15000		—/15000
Remarks	Giddy by swallowing movements during fall							

No. 13.

Rise 200 mm in 20 min. Fall immediately on cessation of rise.

3/5 27 Pt. No. 8	Before experiment	Beginning of rise	7 min. later	15 min. later	20 min. later	Beginning of fall	10 min. later	5 min. down
a_1M	1.0	0	3	6	7	0	0	0
a_1A	30			20	20	30	25	33
Li	19			19	24	24	24	22
Ls	16000			16000	16000		16000	16000
Remarks	÷ giddiness				÷ increased buzzings			

No. 14.

Rise 150 mm in 15 min. Up for 10 min. Fall.

5/5 27 Pt. No. 53	Before experiment	Beginning of rise	10 min. later	15 min. later	Up 5 min.	Beginning of fall	5 min. later	10 min. later	5 min. down	10 min down
a_1M	7	5	8	14	13	6.5	0	1	12	14
a_1A	14	9	10	10	12	12	8	9	15	14
Li	36	36	55	55	55	36	36	36	36	36
Ls	16000	15000		16000	16000	16000		16000		16000
Remarks	÷ giddiness			÷ headache.			Buzzings not increased			

No. 15.

Rise 100 mm in 10 min. Fall immediately on cessation of rise.

10/5 27 Pt. No. 53	Before experiment	Beginning of rise	5 min. later	7 min. later	10 min. later	Beginning of fall	5 min. later	7 min. later	c. 8 min. later	10 min. later	Down	15 min. down	10 min. down
a_1M	7/13	8/11	8/10	8/9	11/15	13/16	8/—	6/9	4/5	3/7	8/8	18/14	18/16
a_1A	12/48	12/—	10/37	9/42	10/42	10/41	7/40	9/38	9/35	10/40	20/67	18/56	18/67
Li	36/21			36/24	36/32	55/24	55/24	—		45/24		36/24	36/21
Ls	16000/16000		16000/16000							15000/—		15000/16000	
Remarks	÷ giddiness. Buzzings unaltered.												

No. 16.

Rise 150 mm in 15 min. Up for 15 min. Fall.

7/9 26 Pt. No. 13	Before experiment	5 min. after beginning of rise	8 min. later	10 min. later	15 min. later	Up 15 min.	5 min. after beginning of fall	10 min. later	15 min. later	10 min. down
a_1M	—/6	—/13	—/7	—/3	—/$2\frac{1}{2}$	—/6	—/3	—/3	—/$2\frac{1}{2}$	—/13
a_1A	—/21			—/22	—/8	—/29	—/16	—/17	—/17	—/20
Li	—/26			—/36	—/55	—/32	—/36		—/45	—/36
Ls	—/16000			—/16000	—/16000	—/17000	—/16000	—/17000	—/16000	—/16000
v.	—/0.50		—/>1.50							
Remarks	Hears better during experiment. No malaise									

No. 17.

Rise 150 mm in 15 min. Up for 15 min. Fall.

7/9 26 Pt. No. 15	Before experiment	5 min. after beginning of rise	7 min. later	10 min. later	15 min. later	Up 15 min.	5 min. after beginning of fall	10 min. later	15 min. later	10 min. down
a_1M	$\frac{}{4\frac{1}{2}}$	$\frac{}{5}$	$\frac{}{7}$	$\frac{}{2}$	$\frac{}{2}$	$\frac{}{10}$	$\frac{}{5}$	$\frac{}{2}$	$\frac{}{3}$	$\frac{}{8}$
a_1A	$\frac{}{17}$			$\frac{}{8}$	$\frac{}{6}$	$\frac{}{17}$	$\frac{}{8}$	$\frac{}{10}$	$\frac{}{11}$	$\frac{}{19}$
Li	$\frac{}{22}$			$\frac{}{36}$	$\frac{}{45}$	$\frac{}{17}$	$\frac{}{32}$	$\frac{}{32}$	$\frac{}{36}$	$\frac{}{22}$
Ls	$\frac{}{17000}$		$\frac{}{16000}$		$\frac{}{16000}$	$\frac{}{20000}$	$\frac{}{16000}$		$\frac{}{18000}$	$\frac{}{19000}$
Remarks	No malaise. Hears better during experiment. Whisper not tested, as it exceded 2.00 before experiment.									

No. 18.

Rise 150 mm in 15 min. Up for 10 min. Fall.

5/5 27 Pt. No. 76	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	Up 5 min.	Beginning of fall	5 min. later	7 min. later	10 min. later	c. 13 min. later	2 min. down	5 min. down	15 min. down
a_1M	$\frac{}{5}$	$\frac{}{5}$	$\frac{}{3}$	$\frac{}{4}$	$\frac{}{5}$	$\frac{}{4}$	$\frac{}{0}$	$\frac{}{3}$	$\frac{}{3}$	$\frac{}{0.0}$	$\frac{}{0.0}$	$\frac{}{8}$	$\frac{}{14}$	$\frac{}{13}$
a_1A	$\frac{}{20}$		$\frac{}{16}$	$\frac{}{12}$	$\frac{}{12}$	$\frac{}{12}$	$\frac{}{9}$		$\frac{}{17}$	$\frac{}{14}$			$\frac{}{20}$	
Li	$\frac{}{30}$	$\frac{}{36}$		$\frac{}{36}$	$\frac{}{36}$	$\frac{}{30}$	$\frac{}{36}$		$\frac{}{36}$	$\frac{}{36}$			$\frac{}{36}$	
Ls	$\frac{}{19000}$		$\frac{}{18000}$		$\frac{}{18000}$	$\frac{}{19000}$	$\frac{}{19000}$			$\frac{}{18000}$			$\frac{}{19000}$	
Remarks	÷ buzzing ÷ giddiness Some pressure on ears													

No. 19.

Rise 150 mm in 15 min. Up for 10 min. Fall.

17/5 27 Pt. No. 14	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	5 min. up	Beginning of fall	2 min. later	3 min later	5 min. later	10 min. later	Down	10 min. down
a_1M	11/8	8/7	8/7	8/7	11/8	12/7	7/6	7/5	6/3½	7/5	5/4	14/9	18/9
a_1A	55/40	45/30	25/27	25/30	30/18	28/18	25/17	35/25		35/25	26/15	40/20	50/28
Li	15/30	36	—	19/30	19/24	—	24			24		19/24	19/24
Ls	16000/16000	16000/16000		16000/16000	17000/17000		17000/17000			17000/17000		17000/17000	17000/17000
Remarks	÷ buzzing ÷ headache ÷ giddiness												

No. 20.

Rise 150 mm. in 20 min. Fall immediately on cessation of rise.

3/5 27 Pt. No. 20	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	20 min. later	Beginning of fall	5 min. later	10 min. later	15 min. later	5 min. down	10 min. down
a_1M	15	10	15	11	14	14	13	8.8	8.8	8.8	20	22
a_1A	32			30	40	32	32	31		25	52	54
Li	24			24	20		24	24		20	15	15
Ls	17000			17000	17000		17000			16500	17000	17000
Remarks	a_1M often 18—20 in good periods. ÷ buzzing ÷ headache ÷ giddiness. Bad period at present											

No. 21.

Rise 150 mm in 15 min. Up for 11 min. Fall.

5/5 27 Pt. No. 66	Before experiment	Beginning of rise	10 min. later	15 min later	5 min. up	Beginning of fall	5 min. later	10 min. later	Down	5 min down	10 min. down	15 min. down
a_1M	13	9	10	11	14	9	7	7	8	22	22	20
a_1A	11	10	11	10	15		12	10		12	12	
Li	30	36	36	36	30		30	36		36		
Ls	16000	15000			16000		16000	16000				
Remarks	a_1M often 20—21 in good periods. Buzzings insignificant, not increased ÷ giddiness ÷ headache											

No. 22.

Rise 150 mm in 15 min. Up for 10 min. Fall.

8/9 26 Pt. No. 3	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	5 min. up	10 min. up	Beginning of fall	5 min. later	5 min. down
a_1M	11	11	10	$8\frac{1}{2}$	12	9.9	11	10	10	16
a_1A	31		25	28	20	25	35		21	32
Li	28		28	32	28	28	32		36	28
Ls	12000		12000		12000		14000			13000
Remarks	a_1M in good periods often = 18.									

No. 23.

Rise 50 mm in 15 min. Up for 10 min. Fall.

17/5 26 Pt. No. 14	Before experiment	Beginning of rise	10 min. later	15 min. later	5 min. up	Beginning of fall	10 min. later	5 min. down
a_1M (10)	5/4	3/3	3/4	5/4	5/4	2/vbr.	0/0	5/4
a_1A (25)	25/25	8/10	10/15	10/15	15/21	12/20	12/25	10/
Li	30/26	36/36		36/36	45/45	45/45	36/36	30/30
Ls	14000/14000		14000/14000		14000/14000		14000/14000	14000/14000

No. 24.

Rise 150 mm in 15 min. Up for 15 min. Fall.

15/9 26 Pt. No. 56	Before experiment	Beginning of rise	5 min. later	10 min. later	Up	5 min. up	10 min. up	15 min. up	Beginning of fall	5 min. later	7 min. later	10 min. later	5 min. down
a_1M	7	7	7	7	2.1	0	5	5	0.0	0.0	1.0	0	7
a_1A	44	32	29	26	35	40	33	42	35	25		24	30
Li	24	40	45		40	28		26	34		36		30
Ls	15000		15000				15000	15000		16000		15500	

No. 25.

Rise 100 min. in 10 min. Fall immediately on cessation of rise.

11/12 26 Pt. No. 63	Before experiment	Beginning of rise	5 min. later	7 min. later	9 min. later	10 min. later	Up 10 min.	Beginning of fall	5 min. later	6 min later	7 min. later	10 min. later	5 min. down
a_1M	9/12	9/9	8/9	8/5	8/5	8/11	9/16	9/13	7/10	5/6	5 vbr.	3 vbr. 4 vbr.	12/16
a_1A	33/54	20/30	22/30			22/30	22/30				25/40	28/41	35/56
Li	28/15	36/24	36/30	36/36			36/34				32/24	24/18	24/18
Ls	17538/16552												
Remarks	Pain in both ears. ÷ giddiness. + unsignificant buzzing												

No. 26.

Rise 150 in 15 min. Up for 15 min. Fall.

14/12 26 Pt. No. 45	Before experiment	Beginning of rise	5 min. later	10 min. later	up	5 min. up	10 min. up	15 min. up	Beginning of fall	5 min. later	10 min. later	12 min. later	Down
a_1M	12/15	4/—	8/—	8/—	11/—	11/—	11.12/—	14/—	8/—	8/—	7/—	6/—	21/—
a_1A	41/57	25/—	25/—	25/—	25/—	30/—	30/—	44/—	22/—	22/—	34/—	34/—	44/—
Li	36/15	55/—		70/—		70/—		36/—		36/—	36/—	36/—	15/—
Remarks	÷ giddiness. Buzzing unaltered. ÷ headache												

No. 27.

Rise 150 mm in 15 min. Up for 15 min. Fall.

5/5 27 Pt. No. 14	Before experiment	Beginning of rise	5 min. later	15 min. later	5 min. up	Beginning of fall	Down	5 min. down
a_1M	5 / vbr.	5 / vbr.	5 vbr. / vbr.	3 / 3	vbr. / 0	0 / 0	2 / 2	4 / 3
a_1A	22 / 15	20 / 15	12 / 12	12 / 12		—		/ 25
Li	36 / 36		90 / 90	45 / 55		55 / 55		36 / 36
Ls	14000 / 14000	14000 / 13000		14000 / 14000		15000 / 14000		14000 / 14000
Remarks	Small attacks of giddiness when swallowing. Buzzing unaltered. Pressure on both ears.							

No. 28.

Rise 150 mm in 15 min. Up for 25 min. Fall.

4/5 26 Pt. No. 14	Before experiment	Beginning of rise	15 min. up	25 min. up	Beginning of fall	15 min. later	20 min. later	10 min. down	1 hour down
a_1M (10)	0 / 0	0 / 0	0 / 0	0 / 0	0 / 0			/ 5	0 / 7
a_1M (22)	3 vbr. / 3 vbr.	3 vbr. / 3 vbr.	3 vbr. / 4	3 vbr. / 4	3 vbr. / 4	5 vbr. / vbr.	vbr. / vbr.	vbr. / 5	vbr. / 11
a_1A (25)	3 / 13	2 / 10	3 / 15	3 / 15	7 / 11	7 / 13	8 / 15	8 / 15	5 / 17
a_1A (105)	6 / 25	0 / 25	9 / 34	0 / 45	12 / 30	/ 30		11 / 33	9 / 45
Li	>150 / 40		>150 / 36	>150 / 32	110 / 32	90 / 34		90 / 34	40 / 20
Ls	15000 / 16000	16000 / 17000	—	16000 / 17000		16000 / 17000			16000 / 18000
v.	0.10 / 2.00		0.20	0.30	0.50				aa / 20.00
Remarks	Some pain in l. ear and a little bleeding in the tympanic membrane after previous experiment with Cordes' apparatus.								

No. 29.

Rise 100 min. in 20 min. Up for 25 min. Fall.

18/5 26 Pt. No. 14	Before experiment	5 min. after beginning of rise	1 min. later	20 min. later	up 15 min.	up 20 min.	up 25 min.	10 min. after beginning of fall	10 min. later	c. 11 min. later	Down	1 hours later
a_1M (10)	5/6	0/0		2/0	0/4	0/0	0/0	0/0	0/0	0/0	4/7	
a_1A (25)	5/22	1/15		2/15	5/15	3/17	3/17	0/15		4/13	15/22	
Li	55/70	>150	>150/40	>150/40	>150/40	>150/26	>150/26	>150/28	150/36		70/24	
Ls	15000/14000		12500/14000	12500/12500	13000/14000		13000/13000	13000/13000			14000/13000	
v.	0.30	0.50	1.00								0.25	aa

No. 30.

Rise 150 min. in 10 min. Up for 20 min. Fall.

7/5 27 Pt. No. 14	Before experiment	Beginning of rise	5 min. later	up	20 min. up.	Beginning of fall	5 min. later	Down	5 min. down
a_1M	5/vbr.	5/vbr.	5/vbr.	4/vbr.	2/vbr.	0/0	0/0	3/vbr.	5/3
a_1A	20/25	7/7	12/10	13/12	12/15	11/12	8/15	12/20	12/20
Li	36/36	45/36		45/36	36/36	24/36	36/36	36/36	36/36
Ls	14000/14000	14000/13000					14000/13000		

No. 31.

Rise 150 mm in 15 min. Up for 10 min. Fall.

21/5 26 Pt. No. 51	Before experiment	Beginning of rise	5 min. later	7 min. later	10 min. later	15 min. later	Up	5 min up	10 min. up	Beginning of fall	5 min. later	7 min. later	10 min. later	5 min. down
a_1M	12	10	11	$9\frac{1}{2}$	$9\frac{1}{2}$	9	8	6	8	7	5	5	6	10
a_1A	27	20	24	22	19	22	22	27	24		24	24	24	28
Li	24		36	50	50	36	36	30					36	26
Ls	8000		8000		9000								8000	8000

No. 32.

Rise 100 mm in 10 min. Up for 5 min. Fall.

22/7 26 Pt. No. 51	Before experiment	Beginning of rise	2 min. later	5 min. later	8 min. later	10 min. later	5 min. up	Beginning of fall	5 min. later	8 min. later	10 min. later	Down	5 min. down
a_1M (10)	$5\frac{1}{2}$ vbr.	7 vbr. 2	7 vbr. 1	5 0	5 0	4 0	6 0	$6\frac{1}{2}$ 0	5 0	5 0	5 0	5 0	4 0
a_1A (25)	7 0	8 0			5 0		5 0	5 0	4 0	4 0	4 0	4 0	4 0
Li	50 >150						50 80	55 80	38 70		38 80	36	36 90
v.	0.10 0			0.35 0			0.30 0		0.35 0			0.75 0	
Remarks	Hears better during experiment. ÷ giddiness. Buzzings not increased.												

No. 33.

Rise 100 min. in 10 min. Fall immediately on cessation of rise.

10/5 27 Pt. No 14	Before experiment	Beginning of rise	5 min. later	8 min. later	10 min. later	Beginning of fall	10 min. later	Down	5 min. down
a_1M	5/2	5/4	5/3	5/4	5/0	0/0	0/0	2/0	5/4
a_1A	17/22	10/12	10/12	10/12	8/12	10/12	8/15		
Li	36/36	45/45	45/45						36/36
Ls	14000/14000	14000/14000					14000/14000		14000/14000

No. 34.

Rise 100 mm in 10 min. Fall immediately on cessation of rise.

13/5 27 Pt. No. 14	Before experiment	Beginning of rise	5 min. later	10 min. later	Beginning of fall	5 min. later	Down	5 min. down
a_1M	4/4	4/4	vbr/vbr	5/4	0/0	0/0	6/0	4/3
a_1A	8/20	7/12	6/8	10/10	15/15			18/25
Li	30/30		36/36	36/36	30/30			30/24
Ls	14000/14000	14000/14000		14000/14000	14000/14000			14000/14000

No. 35.

Rise 150 mm in 15 min. Up for 10 min. Fall.

8/9 26 Pt. No. 55	Before experiment	Beginning of rise	5 min. later	10 min. later	15 min. later	5 min. up	7 min. up	10 min. up.	Beginning of fall	5 min. later	8 min. later	10 min. later	5 min. down
a_1M	$\frac{}{7}$	$\frac{}{4}$	$\frac{}{4}$	$\frac{}{5}$	$\frac{}{5}$	$\frac{}{8}$	$\frac{}{6}$	$\frac{}{6}$	$\frac{}{5}$	$\frac{}{4}$	$\frac{}{3}$	vbr.	$\frac{}{12}$
a_1A	$\frac{}{10}$		$\frac{}{11}$	$\frac{}{11}$	$\frac{}{12}$	$\frac{}{6}$	$\frac{}{11}$	$\frac{}{11}$		$\frac{}{4}$			$\frac{}{11}$
Ls	$\frac{}{8000}$				$\frac{}{8000}$	$\frac{}{8000}$							$\frac{}{8000}$

During the constantly increased pressure boneconduction and airconduction are lengthened, and lower limit falls somewhat, without hearing, however, becoming normal.

After the bell experiment, when the normal atmospheric pressure is again in action, the normal acoustic values again appear.

If the same experiments are made with pts. suffering from otosclerosis nothing happens, bone- and airconduction remain the same as they were before the experiment (see table V pag. 108).

We now come to the question, what factors give rise to the abridgement of boneconduction in normal individuals both on rise and fall of pressure?

In this conjunction regard must first of all be taken to the noise of the engine.

We were originally of opinion that the noise was of no particular importance. But since Prof. T h o r n v a l has substantiated that the noise alone could reduce the boneconduction values about 50 %, we have taken up the influence of noise on the acoustic function for more exhaustive investigation.

The influence of noise on boneconduction. We have made use of a rapidly revolving centrifugal machine with a loud hum. We found that the noise thus produced, like the bell experiment, had an elective influence on the boneconduction of normal individuals.

The degree of abridgement of boneconduction caused by the noise, was different in different individuals. At first sight, it seems probable, that the abridgement of boneconduction in noise, is due to deafening. This cannot, however, be correct, for then the air-conduction would be similarly reduced, which is not the case, as the following example of experiment with the centrifuge proves, a_1M (15) = 5 at same time a_1A (40) = 31.

When pts. with otosclerosis are placed beside the centrifugal machine no change takes place either in bone- or airconduction, although the noise is perceived.

If we start from B e z o l d's theory that boneconduction passes, at least partially, via the labyrinthal wall, ligamentum annulare and the foot plate, and supplement this with our own modification, according to which the conduction by this path depends on the contact between footplate and window frame, it would seem but reasonable to deduce that the reduction of boneconduction, caused by noise, here meets with its explanation. Noise, namely, produces per airconduction an oscillation of the footplate, necessarily causing on each oscillation a lessened contact between the footplate and the labyrinthal wall. This agrees with the fact that pts. with otosclerosis, although they hear the noise, exhibit no abridgement of boneconduction, the fixation preventing the interruption of contact. We also found in one case, where a pt. with otosclerosis could not hear the noise by airconduction (a_1A (40) = 0), that no abridgement of boneconduction ensued (a_1M (15) = 7) even when pt. placed her forehead on the machine, although the noise from it was distinctly perceived per boneconduction when in this position.

In the bell experiments we must therefore take into consideration the fact that boneconduction in normal persons becomes abridged by noise.

Cause of abridgement of boneconduction in the bell experiments. In cases, however, in which boneconduction became nil during fall of pressure, the explanation cannot be sought in the noise alone. There must be another factor present. This cannot be sought in the eventual movements of the tympanic membrane causing tension of the ossicular chain, as that would result in lengthening of boneconduction, (as is also sometimes seen when the individual feels tension in the ears).

In accordance with our general hypothesis, we suppose that the footplate, both during rise and during fall of pressure, is displaced and loses contact with the window frame, the footplate being displaced medially during rise because there is a greater pressure in the middle ear than in the labyrinth, whilst during fall, when there is greater pressure in the labyrinth than in the middle ear, it is displaced in lateral direction. This alteration of the degree of contact between the footplate and the window influences boneconduction, as is evident in these experiments, at least during fall.

That boneconduction is most abridged during the fall of pressure when the footplate is displaced laterally, accords well with B e z o l d's investigations, which prove that the footplate is much more easily displaced in a lateral than in a medial direction. That pts. with otosclerosis, whose stapes are fixed, are not affected, is also comprehensible.

Bell experiments on Menière pts. The findings here — although partially different from the above — seem to corroborate our hypothesis.

35 experiments were made on 24 pts. (7 experiments on No. 14, 3 experiments on No. 31, 2 experiments on No. 53 and No. 29).

Table VI shows the experiments. In each individual case the tables give in detail the methods employed with regard to height of pressure, duration of the constant pressure and fall of pressure.

If we examine boneconduction during the bell experiments, we find that exps. 1—8 exhibit exactly the same conditions as in normal individuals. Boneconduction was abridged during rise and during fall. In exps. 9—15 boneconduction is lengthened, in spite of the noise, during the whole rise, and abridged during the fall. In exps. 16—17 boneconduction is lengthened in spite of the noise and decreases afterwards during rise of pressure, then decreases evenly during the fall. In the rest of the exps., from 18—35, boneconduction remains fairly unaltered during rise of pressure, in spite of noise, and then decreases during the fall except exps. 22, 28, 32.

In exps. 1—8, as above stated, we find boneconduction abridged, during rise of pressure, exactly as in normal individuals. These 8 pts. were examined during good periods, when they felt well. Nos. 57 and 47, indeed, considered themselves perfectly well. It may therefore be supposed that there was nothing, either in the

middle ear or labyrinth, which could prevent the medial displacement of the footplate.

In exps. 9—15 we find boneconduction constantly lengthened — in spite of the noise. Here we must suppose that the footplate moves in a medial direction, endeavouring to attain an ideal contact to the labyrinthal wall, without being able to pass this and become further displaced, as in normal individuals.

In exps. 16—17 we meet with first a lengthening of boneconduction during rise, attaining in Pt. No. 13 normal value, and then a diminishing. This must mean that the displaced footplate has moved in a medial direction during rise of pressure, reached a lateral position to the labyrinthal wall and then moved on in a medial direction, which latter circumstance shows itself by an abridgement of boneconduction. In these experiments, in contradiction to what happens in exp. 9—15, the footplate must have overcome resistance.

In exps. 18—35 we find that in some cases boneconduction remains almost unaltered during the rise of pressure, in others it varies a little without exhibiting any decided type. The footplate probably swings backwards and forwards, vainly endeavouring to overcome the resistance and attain a more medial position. Thus with the exception of exps. 1—8, which were made on pts. in exceptionally good health, all the Menière pts. exhibited abnormal conditions of boneconduction in the bell experiments and this, in our opinion, finds its explanation in the hypothesis, that there is in all these cases a certain factor, which in a greater or lesser degree hinders stapes in moving in a medial direction during the rise of pressure.

Nearly all the Menière pts., on the other hand, exhibited the same conditions as normal individuals during fall of pressure; that means to say, that there is no hindrance to the lateral movement.

At the termination of the bell experiments the boneconduction of as good as all Menière pts. is much longer than it was before the experiment, for inst. Pt. No. 29 expm. No. 8: prior to 5 after 11, Pt. No. 43 expm. No. 5: 7—10, Pt. No. 36 expm. No. 3: 6—10, Pt. No. 57 expm. No. 1: 12—21, Pt. No. 53 expm. No. 15: 7—18, Pt. No. 13 expm. No. 16: 6—13, Pt. No. 15 expm. No. 17: 4½—8, Pt. No. 20 expm. No. 20: 15—22, Pt. No. 66 expm. No. 21: 13—20,

Pt. No. 45 expm. No. 26: 12—21, Pt. No. 55 expm. No. 35: 7—12, Pt. No. 76 expm. No. 18: 5—13. This might indicate, that the labyrinthal fluid had been somewhat displaced and during the fall of pressure had not quite returned to its position, the footplate thus being closer to the window frame, eventually in an ideal position for maximal boneconduction, instead of being laterally displaced, as was formerly the case. The lengthening of boneconduction may be maintained for several hours.

This agrees with Chilow's experiments. On examining labyrinthal pressure in animals by means of a manometer in the labyrinth, he has found that increased pressure in the external meatus makes the fluid in the manometer rise some millimeters, whilst succeding secession of pressure in the external meatus does not make the fluid sink to its original level.

The same regular rise of lower limit, which we observed in our experiments on normal individuals both during rise and fall of pressure, was not present in our Menière pts. On rise of pressure 26 cases exhibited rise, 2 cases (exps. 12, 20) fall, whilst 6 remained unaltered. The great majority of cases exhibited a fall of lower limit during the fall of pressure, indeed to such a degree that lower limit was better than it was before exp. (exps. 1, 3, 5, 8, 11, 12, 19, 20, 25, 26, 28, 29, 32); in 9 exps. (10, 13, 17, 21, 22, 23, 24, 27, 31) lower limit was raised, — but only a little; in the remainder it continued unaltered.

Starting from our idea, that a fixation of the ossicular chain causes a rise of lower limit, the improvement of lower limit, as exhibited by these pts. during fall of pressure, must depend upon the occurance of something causing a diminished fixation of the ossicular chain.

Airconduction was inconsiderably abridged in the majority of cases, both during rise and fall of pressure; in a number of cases it remained unaltered. It is thus evident that airconduction is not nearly so abridged as in normal individuals. In 4 exps. (1, 9, 20, 28) it was even distinctly improved during the rise, in 8 exps. (3, 15, 16, 21, 27, 28, 32, 35) unaltered.

This circumstance must be interpreted in the same way as the conditions relating to lower limit, i. e. as a result of the ossicular chain not becoming so firmly fixed as in normal individuals.

In a few cases (exps. 10, 11, 16, 28, 29) we were able to test hearing of whisper, as it was so defective that the dimensions of the bell were sufficient. The hearing of whisper increased during the fall in exps. 10, 11, 16; in exps. 28 and 29 not before the cessation of fall.

The improvement in hearing of whisper must be interpreted in the same way as the improvement of lower limit, namely by a lessened fixation of the ossicular chain and of the footplate.

The bell experiments, as all the other cited experiments and clinical observations, can thus be explained by the hypothesis here set up.

The influence of dislocation, abnormal mobility and fixation of the footplate. Our Menière pts., as said before, exhibited various types of boneconduction.

In such cases where there was a disproportional abridgement of boneconduction, we must suppose, that the footplate is dislocated laterally. An abnormal mobility of the footplate would be favourable to such a dislocation. We also understand that the footplate is able to change its position in relation to the window-frame. The result is, that boneconduction values, as we have shown, can change spontaneously in experiments with fluid metabolism and in bell experiments.

The lateral dislocation of the footplate need — provided the footplate is not hampered in its movements (fixed) — only manifest itself by abridgement of boneconduction, which is in accordance with the cases mentioned in literature, (B l u m e n t h a l, H a m m e r s c h l a g), also with our own cases of abridged boneconduction and normal hearing of whisper, normal airconduction and normal lower limit. (Nos. 15, 35, 56, 108, 122, 123, 128).

But, provided the dislocated footplate also becomes hampered in its movements, that is to say becomes fixed, it will, besides by abridged boneconduction, be acoustically evinced by an abridged airconduction and raised lower limit, also by diminished hearing of whisper. The reduction of hearing is an expression of the degree of fixation.

Fixation may be *temporary* or *permanent.*

If fixation is removed hearing will again improve, as will be seen by a better hearing of whisper, longer airconduction and sinking of lower limit; hearing may fluctuate.

When fixation is permanent it will acoustically evince itself by a constantly impaired hearing of whisper, constant abridgement of airconduction and raised lower limit. If this condition continues long enough, secondary changes will appear in the middle ear, analogous to those we meet in other parts of the body when a joint becomes immobile.

Localization of the causative morbid condition. The cause of the primary fixation is supposed to be a temporary or permanent incongruity between the pressure in the labyrinth and the pressure in the middle ear.

It would seem reasonable to first of all examine whether the cause of the changing hearing exhibited by our pts. is to be sought for in the varying labyrinthal pressure. The facility with which hearing changes proves, that if this is the case we also have to do with an easily altered labyrinthal pressure.

The influence of lumbal puncture on hearing. In literature we find statements showing that lumbal puncture is effective against giddiness and eventually gives an improvement of hearing, diminished buzzings and less headache. Babinski in 32 cases of giddiness attained improvement in 21, and in the 7 cases the improvement lasted for over 6 months. Bárány performed lumbal puncture in 7 cases of »stasis in the pontine cysterne» with good results. Holmgren has 3 cases in which this operation gave relief. James Putnam and H. Brunner[2] also recommend lumbal puncture. In cases of syphilis O. Beck[2] has, as before mentioned, performed lumbalpuncture and proved that a lengthening of boneconduction ensued. Grahe[1] recommends lumbalpuncture for pts. with nephritis, whose hearing is impaired. In all these cases we must suppose that lumbalpuncture acted beneficially on the labyrinthal pressure and, therefore also on the function of the labyrinth.

Relation between the attacks of giddiness and the fluctuating hearing. Many pts., as above mentioned, had an improvement of hearing in sympathy with their attacks of giddiness. This may possibly be connected with the increased loss of fluid, as vomiting and diarrhoea appear at the same time, and, now and then, vasomotor rhinitis. Besides at such times the pt, takes no nourishment. A few have also remarked greater diuresis during,

or immediately after, the attacks (Nos. 14, 16, 85). Others have noticed perspiration, salivation, epiphora (for inst. Nos. 25, 43, 55).

This might indicate that the fluid contents of the labyrinth and therewith the tension medial to the footplate, was diminished, by which the fixation of stapes would be done away with.

In the cases where hearing becomes worse after an attack of giddiness, we must suppose that the attack of giddiness is the result of increasing labyrinthal pressure, and that this has caused a fixation of the footplate. In some cases the cause may wholly, or partly, be a decreasing pressure in the middle ear resulting from resorbtion of air here on account of tubal stenosis.

The varying degree of the footplate's fixation may explain the changing hearing. A sudden mobilisation of the footplate will explain the sudden attack of giddiness.

In a single case (No. 14) we were able to test the acoustic function 1 hr. before an apoplectiform attack, and again immediately after it.

$$14/5\text{:}\quad v\,\frac{aa}{0.30}\;a_1M\;(10)\,\frac{0}{0}\;a_1A\,(25)\,\frac{4}{6}\;Li\,\frac{>150}{>150}$$

$$1\text{ hr. later:}\quad v\,\frac{aa}{20.00}\;a_1M\;(10)\,\frac{0}{5}\;a_1A\,(25)\,\frac{4}{19}\;Li\,\frac{>150}{36}$$

This great difference does not seem explicable otherwise than by supposing that the footplate has suddenly been released from fixation on the inner side. This reminds us that Lucae[7] has observed a pt. with attacks of giddiness, abridgement of bone-conduction and v = 0.30, whose hearing improved so much after treatment with the pressure probe, that on the next day v was 10.00—13.00.

In this connection we may mention that Gellé[4], Thost[2], Robinson, Lucae[5], F. Alt[3] and E. Ruttin speak of attacks of giddiness in connection with catheterization and with tubal stenosis, and Alt[3] distinctly points out that giddiness from catheterization is not due to a caloric reaction, as Bárány[2] thinks, but to the rise of pressure, as giddiness never shows itself when only a slight pressure is used for the inflation. Many authors have observed that Menière pts. often get their attacks after catheterization, which is therefore disadvised. As before mentioned,

many of our pts. became giddy during catheterization, a few had violent attacks immediately after it. Gellé[4] speaks of giddiness from compression of the external meatus. Müller mentions that giddiness may arise from pressure on stapes and disappear again on rarefaction of air in the external meatus. Löwenberg mentions giddiness after blowing the nose and after catheterization. Bouchut mentions giddiness after sneezing and observes, that the giddiness may be avoided by pressing the ears. G. Heermann, in his examination of caisson labourers mentions that 3 labourers, who had had Menière attacks after coming up from the bell and who had felt unsteady for several days after, felt quite well again when they were once more brought into overpressure. Heermann points out that this is a good argument against the supposition that the attacks are the result of hemorrhage or gasembolia, as Heller, Mager and v. Schrötter have supposed.

It would therefore seem reasonable to suppose, that it is the movements of the fenestrae, especially the fenestra ovalis, which cause the sudden attacks of giddiness. It seems likely, that in the cases mentioned in literature as also in the pts. in our material, we are to suppose that stapes is abnormally mobile, just on account of a frequently changing labyrinthal pressure; the ligament of the footplate presumably becoming too highly stretched during the increased pressure. Such an abnormal mobility will also continue after the cessation of the abnormal pressure.

In favour of an abnormal mobility is also the circumstance that some of the pts. (Nos. 2, 6, 14, 53, 58, 83, 88) exhibited Hennebert's fistula symptom; No s. 100, 132 besides had a trace of the same, as they stated that in bad periods they could not touch their ear without feeling giddy.

Rob. Lund[2] has explained Hennebert's fistula symptom in pts. with congenital syphilis as being the result of abnormal mobility of the footplate, (resulting from a gumma in the neighbourhood of the fenestra), whereas he, strange to say, in nonsyphilitic cases seeks the cause in abnormal central conditions (Lund[3]).

That the mobility of the fenestra causes the giddiness is also in accordance with the circumstance, that when Menière pts.' hearing is constantly bad, i. e. the footplate permanently fixed,

the sudden attacks of giddiness cease, a thing so well known that the medical man often comforts his pt. by saying »the attacks of giddiness will disappear when hearing is lost».

All these phenomena seem to us a confirmation of the justness of our hypothesis that it is the changing labyrinthal pressure which produces the attack, and that the more violent the change, the more violent the attack. The slighter attacks, which frequently accompany bad hearing and most frequently show themselves in unsteadiness, depend presumably on the lesser changes in the labyrinthal pressure. Thus we can understand that a severe attack may »make up for» several smaller ones, and by so doing bring about a longer period of immunity.

The acoustic findings and the relation between the fluctuating hearing and the attacks of giddiness can thus, in all their variations be interpreted as the result of an affection of the sound conducting apparatus.

This opinion, that in mb. Menièri we have to do with an affection of the sound conducting apparatus, is further borne out by the otoscopical alterations and the state of tuba.

To this we may add, that the autophonia and diplacusis which was observed in many of our pts. must be taken as evidence of a disease in the sound conducting apparatus, as all authors, according to K. G r ü n b e r g, are unanimous in interpreting autophonia as a sign of a tuba — middle ear disease. Diplacusis is generally considered as being much more frequent in sound conducting troubles than in perception troubles.

Conclusion.

All the above mentioned clinical and experimental acoustic phenomena found in pts. with mb. Menièri may then be explained from the point of view of a changing, at times increased, labyrinthal pressure. The increased pressure will fix stapes laterally to the lateral labyrinthal wall, and find its acoustic expression in a reduced hearing of whisper, abridged airconduction, altered boneconduction and raised lower limit. As soon as the pressure decreases fixation will decrease, this will show itself by increasing hearing of whisper, longer airconduction, altered boneconduction and improved lower

limit. In case there is a constant increase of labyrinthal pressure and abnormal fluid content in the labyrinth, the fixation of stapes will continue unaltered. Besides the characteristic picture of a sound conducting affection, secondary degeneration of the sensory epithelium or of the nerve may then appear, expressing itself by the lowering of upper limit.

The vestibular phenomena in mb. Menièri seem to find a natural explanation in the changing labyrinthal pressure, in accordance with S. H. Mygind's[2] hypothesis as to the vestibular function of the labyrinth.

The next problem is, in what does the increased labyrinthal pressure consist and what is it that causes alterations of the pressure?

It may be a question of a hydrops labyrinthi, which Wittmaack[1], on the ground of experimental and pathological-anatomical investigations, supposes to be the basis of a series of labyrinthal diseases. This opinion is supported by many authors. Perhaps it is a question of an oedema of the labyrinth, analogous to Quincke's oedema, as Quincke himself suggested. It is presumably a question of vasomotor disturbances of a both more universal and local nature, in the form of spasms and dilatation of the vessels such, as reported by numerous authors (Lermoyez, H. Brunner[2], Kobrak[1, 2, 3], Szász[2], Trautmann, Démétriades). In a series of our cases it is tempting to draw comparisons between tubal stenosis, vasomotor rhinitis (56 of 89 examined exhibited tubal stenosis, 29 vasomotoric rhinitis) and anomalities in fluid metabolism, which so often appear in connection with aural phenomena. According to this we should, in these cases, have to do with an oedema in the nose, the tuba and also the labyrinth. However we imagine these processes in the labyrinth, they will all be capable of resulting in an increased changing labyrinthal pressure.

A real solution of the problem here sketched out can, however, only be arrived at through a general medical consideration of the subject. If we examine our pts. more closely we shall find that it is remarkable how they nearly all exhibit symptoms of a more universal trouble: 115 suffered from headache, 53 from rheumatism, 46 from gastro-intestinal troubles, 66 from subcutaneous — often very considerable — infiltrations, further many are chilly, suffer from

cold extremities, numb fingers, cyanosis and congestions, all expression of a dysregulation.

The salyrgan experiments also point in the same direction. According to Poul Saxl and Rob. Heilig's experiments, increased diuresis, at least in experiments with novasurol, appears as a rule, only in those cases in which there is oedema. Knud Møller believes that he is justified in declaring, that although it is true that an increase in diuresis takes place in normal persons after treatment with salyrgan, this increase is a few hours later on compensated for by a decrease, the diurnal diuresis thus remaining practically unaltered, while the daily diuresis of pts. with hydropsis is greatly increased. It was further shown by Møller's experiments on rabbits, that an intake of salyrgan in doses corresponding to those generally given in treatment of oedema, gave no increase in the diuresis unless the animal experimented on had been given a great quantity of water.

Our pts. must, provided the above is correct, have had an abnormal fluid metabolism, a universal abnormal deposing of fluid in the organism, as the majority of them, in spite of a previous »draining» exhibited considerable increase in diuresis after salyrgan without any compensatory decrease following.

To unravel all these conditions it would therefore be necessary, among other things, to more closely investigate fluid metabolism and, not least, the function of the capillaries, with a special consideration of the fundamental views advanced by August Krogh.

All this is, however, outside the scope of the present work. Our aim has only been to investigate what can be deduced from the acoustic phenomena — especially boneconduction — with regard to ear disease in mb. Menièri.

English Summary.

By mb. Menièri we understand a disease which as far as the ear is concerned, is characterized by changing acoustic and vestibular abnormalities, and which has no specific etiology.

Our material consists of 135 pts. (70 males and 65 females) of age varying between 14 and 74.

In the beginning of the disease hearing of whisper may be normal (ɔ: $>$ 20 m.). Even in advanced cases with considerable hardhearing during bad periods, the hearing of whisper may during following good periods, be restored to the normal.

Airconduction exhibits corresponding fluctuations, except that, even in good periods it is but exceptionally restored to the normal.

Lower limit is nearly always elevated, especially during bad periods.

Upper limit is as a rule normal, with an tendency to becoming impaired in advanced cases. Sometimes it rise paradoxically when the hearing, as determined by the other tests, is reduced, and is again lowered when the other qualities are improved.

Boneconduction in some cases is lengthened or normal, generally however it is abridged, often to a much greater extent than airconduction, or is nil.

Hearing as a whole, is markedly fluctuating, sometimes reaching from nearly perfect deafness to practically normal function.

The acoustic function is generally affected on both sides.

Hearing often changes in sympathy with an attack and may as often increase as decrease.

Buzzings exist in the great majority of cases (85 $^0/_0$), often on both sides; their intensity varies greatly in the same patient.

All our patients exhibit nystagmus abnormalities.

The great majority exhibit abnormalities of the tympanic membrane.

Tubal stenosies are frequent.

As other signs of middle ear affections autophonia and diplacusis may be mentioned and sensation of pressure on the ears.

Hearing is influenced by water metabolism.

This may be experimentally proved by reducing the salt and the water intake of the patients, or by giving them various diuretics.

This was particular evident by salyrgan (77 experiments) where an increase of the diuresis, together with loss of weight and thirst, may appear in sympathy with a considerable increase of hearing, in course of some hours.

The effect on hearing by pilocarpin, thiosinamin, fibrolysin probably depends on a corresponding influence on water metabolism.

On the other hand an increased intake of fluid together with an increase of weight may reduce hearing.

The same alterations of water metabolism will explain a series of clinical observations of fluctuating hearing (relative to time of the day, exercise, degree of moisture of the air, diet, baths, oedemata).

The experimentally produced changes of hearing exhibit the same characteristic features as the spontaneous fluctuations with regard to all the details of the single acoustic tests.

The elevated lower limit indicates an affection of the sound transmitting apparatus.

The generally well preserved upper limit speaks against a presumption of an affection of the sound perceiving apparatus.

There is an unanimous opinion that a lengthening of boneconduction as compared to airconduction is due to an affection of the sound transmitting apparatus.

Logically speaking the same must be the case as far as the abridgement of boneconduction as compared to airconduction.

To explain an abridged boneconduction resulting from an affection of the sound transmitting apparatus the following hypothesis is suggested as a further development of Bezold's theory of boneconduction.

The boneconduction sound is transmitted to the endolymph partially via labyrinthal wall — ligamentum annulare — footplate, partially via tympanic membrane — ossicular chain — footplate. If transmission along any of these two pathways is altered, boneconduction will also alter.

If the isolation of the ligamentum annulare becomes injured boneconduction will be improved (as in otosclerosis) on account of a more intimate contact between labyrinthal wall and footplate. Consequently a reduction of this contact must result in an abridgement of boneconduction. Such conditions may arise by a dislocation of the footplate.

If the ossicular chain becomes fixed a lengthening of boneconduction will also appear. A lengthening on this account and an abridgement on account of stapes dislocation may eventually counteract each other, resulting in apparently normal boneconduction although in reality the sound transmitting apparatus is impaired.

By this hypothesis we are able to explain that a manipulation of the sound transmitting apparatus may result in an abridgement as well as in a lengthening of boneconduction as seen from experiments made by Bezold, Mach, Gellé and others.

The same is the case in water and quicksilver loading, experiments performed by the author in patients radically operated upon.

The elective abridgement of boneconduction in noise which appears in normal individuals, but is absent in patients with stapes anchylosis, may be explained, in conformity with the above theory as depending upon a reduction of contact between labyrinthal wall and footplate caused by the noise, transmitted per airconduction, making the footplate vibrate.

Further experiments have been made in an overpressure-chamber (»the bell»). Here boneconduction in normal individuals becomes abridged during rise, and particularly during fall of pressure. Airconduction is comparatively less impaired and is perhaps best during fall. In cases of stapes anchylosis boneconduction remains unaltered during these experiments. Several Menière-patients with a previously abridged boneconduction may exhibit lengthened or unaltered boneconduction during rise; during fall boneconduction always becomes further abridged.

Although the influence of the noise made from the pump of the chamber cannot be excluded, the above experiments seem only capable of explanation by supposing a changing contact between labyrinthal wall and footplate by a dislocation of the footplate on account of the difference between the pressure in the middle ear and the labyrinth.

In the same way the abridged boneconduction in Menière patients is due to a laterally dislocated footplate.

Elevation of lower limit, abridgement of airconduction and impaired hearing of whisper depends on a fixation of the footplate.

The fixation and the lateral dislocation of the footplate is caused by an increased, but as the changing acoustic function shows, fluctuating pressure in the labyrinth, eventually by a decrease of pressure in the middle ear (tubal stenosis) or by both factors acting together.

Secondary fibrous changes may make the stapes fixation permanent. Secondary nervous degenerations on account of the increased endolabyrinthine pressure will affect upper limit.

The increased endolabyrinthine pressure, eventually combined with secondary nervous degenerations, explain the vestibular phenomena.

The apoplectiform attacks of giddiness with sudden change of boneconduction are caused by a sudden displacement of the stapes.

The abnormal mobility of the footplate explains the fistula symptom of Hennebert, found in 7 pts., and the giddiness and nystagmus so often observed by inflation of air into the middle ear.

The reason of the increased and changing endolabyrinthine pressure is presumably to be sought in abnormal vasomotor conditions in the labyrinth.

The many pathological phenomena of a more universal character, which will always be found present in Menière patients and which also may be brought in relation to water metabolism seem to agree in favour of the above hypothesis.

Danish Summary.

Ved Mb. Menièri forstaar vi en Lidelse, der for Ørets Vedkommende viser sig i vexlende akustiske og vestibulære Abnormiteter, og som er uden specifik Ætiologi.

Vort Materiale bestaar af 135 Pt. (70 Mænd + 65 Kvinder) i Alderen mellem 14 og 74 Aar.

I Sygdommens Begyndelse kan Hørelsen for Hviskestemme være normal (ɔ: > 20 mtr.). Selv i fremskredne Tilfælde med betydelig Tunghørighed i daarlige Perioder kan Hørelsen for Hviskestemme i de gode Perioder være normal.

Luftledning viser tilsvarende Svingning, er dog selv i gode Perioder kun undtagelsesvis helt normal.

Nederste Grænse er næsten altid oprykket, især i daarlige Perioder.

Øverste Grænse er som Regel normal med Tendens til Nedrykning i de fremskredne Tilfælde. Undertiden viser den et paradoxalt Forhold ved at være stigende, naar Hørelsen er daarligst, og faldende, naar Hørelsen er bedst.

Benledningen er i enkelte Tilfælde forlænget eller normal, men som oftest er den forkortet og ofte mere end svarende til Luftledningen, ikke sjældent er den helt ophævet.

Hørelsen som Helhed er stærkt skiftende, sommetider svingende fra omtrent komplet Døvhed til praktisk talt normal Hørelse. Den akustiske Funktion er som Regel dobbelsidig afficeret.

Hørelsen skifter ofte i Tilslutning til Svimmelhedsanfald, kan blive saavel bedre som daarligere.

I de fleste Tilfælde er der Øresusen, ofte dobbeltsidig og meget vexlende hos samme Patient. Øresusen manglede i 15 %.

Alle vore Patienter frembyder Nystagmusabnormiteter.

Langt de fleste frembyder Trommehindeforandringer.

Tubastenoser er hyppige.

Af andre Symptomer paa Mellemørelidelse, som af og til findes, kan nævnes Autophoni og Diplacusis og Tryk for Ørerne.

Hørelsen er afhængig af Væskestofskiftet.

Dette er experimentelt paavist ved at indskrænke Patienternes Væske- og Saltindtægt samt ved at behandle dem med forskellige Diuretica.

Resultatet er særlig tydeligt ved Indgift af Salyrgan (77 Forsøg), hvor en forøget Diurese med Vægttab og Tørstfølelse kan give Hørelsesforbedring i Løbet af nogle Timer.

Den forbedrede Hørelse ved Brug af Pilocarpin, Thiosinamin, Fibrolysin skyldes antagelig ogsaa en Indvirkning paa Væskestofskiftet.

Omvendt viser det sig, at en forøget Væskeindtægt med Vægtforøgelse giver nedsat Hørelse.

En Række kliniske Iagttagelser af vexlende Hørelse lader sig ligeledes forklare ud fra et svingende Væskestofskifte (f. Eks. Hørelsessvingning afhængig af Ødemer, Bade, Diæt, Motion, Døgnsvingninger).

Den experimentelt fremkaldte Hørelsessvingning viser de samme karakteristiske Forhold som den spontane Hørelsessvingning med Hensyn til de enkelte akustiske Funktionsprøver.

Den oprykkede nederste Grænse tyder paa en Lydledningslidelse.

Den som Regel velbevarede øverste Grænse taler imod en primær Lidelse af det lydperciperende Apparat.

Der er almindelig Enighed om, at en i Forhold til Luftledningen forlænget Benledning, er Udtryk for en Lydledningslidelse. Heraf følger logisk, at det samme ogsaa maa være Tilfældet med en uforholdsmæssig forkortet Benledning.

For at forklare, hvorledes en forkortet Benledning kan være Udtryk for en Lydledningslidelse, opstilles som Udvidning af Bezolds Benledningstheori følgende Hypothese.

Benledningstonen overføres til Endolymphen dels via Labyrinthvæg — ligamentum annulare — Fodplade og dels via Trommehinde, Ørekædeknogle og Fodplade. Sker der Forandring af Ledningsevnen paa een af disse 2 Veje, maa Benledningen forandres. Hvis Isolationen ved ligamentum annulare nedbrydes, vil Benledningen forlænges (som ved Otosclerose) paa Grund af en mere intim Kontakt mellem Labyrinthvæg og Fodplade. Omvendt maa følgelig en Formindskelse af Kontakten her give en forkortet Benledning. Et saadant Forhold vil kunne fremkomme ved en Dislocation af Fodpladen.

Hvis Øreknoglekæden fixeres, vil Benledningen blive forlænget. En saadan Forlængelse og en Forkortning paa Grund af Stapesdislocation vil eventuelt kunne modvirke hinanden og resultere i en tilsyneladende normal Benledning trods bestaaende Lydledningslidelse.

Ved Hjælp af denne Hypothese er vi i Stand til at forklare, at Paavirkning af det lydledende Apparat kan give saavel forkortet, normal som forlænget Benledning, saaledes som vist af B e z o l d, L u c a e, M a c h, G e l l é og andre gennem talrige Experimenter.

De samme vexlende Benledningsværdier paavist af Forfatteren ved Forsøg med Vand og Kvægsølvbelastning i Øregangen hos radikaloperede lader sig ligeledes forklare ud fra ovenfor opstillede Hypothese.

Den elektivt forkortede Benledning, som ved Støj viser sig hos normale, men ikke hos Otosclerosepatienter, lader sig ligeledes forklare gennem den opstillede Theori, som beroende paa en formindsket Kontakt mellem Fodplade og Labyrintvæg fremkaldt ved, at Støjen pr. Luftledning sætter Fodpladen i Svingninger.

Der er tillige gjort Experimenter i Overtrykskammer »Klokken». Benledningen forkortedes her hos normale saavel under Trykstigning som under Trykfald, men stærkest under Faldet. Luftled-

ningen forkortedes forholdsvis mindre. Ved Stapesankylose var Benledningen uforandret. En Del Menièrepatienter med en i Forvejen kort Benledning fik uforandret eller forlænget Benledning under Stigningen, men yderlig forkortet Benledning under Faldet.

Selv om Støjen fra Maskinen i »Klokken» maa tages i Betragtning, maa Experimenterne forklares ud fra Antagelsen af en skiftende Kontakt mellem Fodpladen og Labyrintvæggen, en Dislocation af Fodpladen opstaaet ved Forskellen mellem Trykket i Mellemøret og Labyrinthen.

Paa samme Maade forklares Menièrepatienternes korte Benledning, som beroende paa en lateral Dislocation af Fodpladen.

Den oprykkede nederste Grænse, den forkortede Luftledning og nedsatte Hørelse for Hviskestemme skyldes en Fixation af Fodpladen.

Fodpladens Fixation og den laterale Dislocation skyldes et forhøjet, men som den vexlende akustiske Funktion viser, skiftende Labyrinttryk eventuel i Forbindelse med et formindsket Tryk i Mellemøret (Tubastenoser) eller begge Dele i Forening.

Sekundære fibrøse Forandringer kan resultere i blivende Stapesfixation.

Sekundær Nervedegeneration som Følge af det forhøjede endolabyrinthære Tryk — vil bevirke Nedrykning af øverste Grænse.

Det forøgede endolabyrinthære Tryk eventuelt i Forbindelse med sekundær Degeneration af Neuroepithelet betinger de vestibulære Fænomener.

De apoplektiforme Svimmelhedsanfald med pludselig Skiften af Benledningen, kan forklares som beroende paa en pludselig Stapesforskydning.

Den abnorme Mobilitet of Fodpladen forklarer Henneberts Fistelsymptom, der paavistes hos 7 Patienter, samt den ved Luftdouche hyppigt optrædende Svimmelhed med Nystagmus.

Aarsagen til det forhøjede skiftende endolabyrinthære Tryk er antagelig at søge i abnorme vasomotoriske Forhold i Labyrinthen.

De mange pathologiske Fænomener af mere universel Art, som saa ofte findes hos Patienter med Mb. Menièri, og som ogsaa muligvis staar i Forbindelse med Væskestofskiftet, taler ligeledes til Gunst for den opstillede Hypothese.

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